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DEVELOPMENT OF A MODEL FORECAST OUTBREAKS OF ENTOMOSPORIUM LEAF AND BERRY SPOT IN SASKATOON ORCHARDS AND PHENOLOGICAL DEVELOPMENT

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Development of a Model to Forecast Outbreaks of Entomosporium Leaf and Berry Spot in Saskatoon Orchards and Phenological Development of the Saskatoon for more Effective Crop Management

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1. ABSTRACT/SUMMARY

Entomosporium leaf and berry spot disease is an important factor limiting fruit yield in saskatoons. In order to improve the quality and quantity of saskatoon fruit yield, phenological and disease forecasting models based on orchard microclimate were created. Temperature, leaf wetness, and precipitation data used in the models were collected using orchard weather stations. A heat unit model for predicting the dates of flowering, fruit harvest and the cessation of axillary vegetative shoot expansion from the date of bud break was created for saskatoon using critical temperatures for plant growth and development. When historical heat unit data were incorporated with actual heat unit data, it was possible to predict the dates of flowering and fruit harvest before they occurred in the field. A dynamic disease-forecast model was created to estimate disease pressure based on the relationship of disease to inoculum potential and production, inoculum release, leaf wetness duration and temperature, and host susceptibility. To make the dynamic disease-forecasting model useful for controlling disease in the field, control thresholds together with application and control guidelines for Topas 250E were added to the model. When tested in 2002 at an orchard near Carman, Manitoba, disease was controlled using two fungicide applications as recommended by the model.

2. EXECUTIVE SUMMARY

The objectives of this project were: a) to determine the microclimatic factors that promote the development of *Entomosporium* leaf and berry spot in saskatoons and phenological development in the saskatoon; b) to establish a relationship between

phenological development of the saskatoon plant and development and proliferation of *Entomosporium* leaf and berry spot; and c) to develop an effective model to forecast outbreaks of *Entomosporium* leaf and berry spot disease, but which may also be adapted for the more accurate prediction of the developmental growth stages of saskatoons. A precise and reliable phenological forecasting model would help to make saskatoon fruit production more efficient by allowing growers to organize pesticide application and fruit harvest operations in advance.

In this study, a model was developed to predict the dates of flowering, fruit harvest, and the cessation of axillary vegetative shoot expansion. The minimum, optimum, and maximum temperature parameters of the potato physiological day model were modified to create a saskatoon physiological day (SASK-Day) model, which utilizes temperatures that are critical for saskatoon plant growth and development. The SASK-Day model was tested against the growing degree-day and Julian day systems for predicting saskatoon growth and development at 2 locations in Manitoba over a 3-year period. Compared to the predictive ability of the growing degree-day and Julian day models, the SASK-Day model more accurately predicted the dates of flowering (±2.1 d), fruit harvest (±2.8 d), and the cessation of axillary vegetative shoot expansion (±5.1 d) from the date of bud break. The saskatoon physiological day model was eventually integrated into a dynamic disease-forecasting model that suggests timely control of entomosporium leaf and berry spot (ELBS) disease using the fungicide Topas 250E.

The influence of leaf wetness duration and temperature on the development of *Entomosporium mespili* (D.C.) Sacc. on *Amelanchier alnifolia* Nutt. (saskatoon) was quantified in controlled-environment studies. Plants were inoculated with a conidial suspension and then subjected to a period of leaf wetness (0, 6, 12, and 24 h) at a constant temperature of 18°C, after which they were moved to a growth room set at a fixed temperature (10, 15, 20, 25, and 30°C) for a period of 14 d. Disease development increased as the leaf wetness duration increased for combined temperature treatments up to 24 h. Temperatures between 10 and 20°C promoted optimal disease symptom development for leaf wetness periods longer than 6 h. Above the optimal temperature range, mean disease levels decreased. ANOVA with orthogonal contrasts and stepwise regression were used to create a regression equation that describes the relationship of *E. mespili* disease development on saskatoon to both leaf wetness duration and temperature. Combinations of leaf wetness periods between 6 and 24 h and temperatures between 10 and 25°C were more optimal for lesion development on younger saskatoon leaves.

It was found that there is a positive linear relationship between the percent of saskatoon leaf area infected (X) and the relative concentration of *E. mespili* conidia (Y) released from that tissue, as represented by the equation ($Y = 5.34\text{x}10^{-2} * X$). In a field study, initial disease symptoms appeared 5 d after the first rainfall event that occurred 1 d or more after the date of flowering. A splash-dispersed conidia trap was constructed and used to trap conidia within the canopy of a saskatoon plant, and in both years of this study, conidia were first trapped on Julian day 184, which coincided with the start of exponential percent leaf area infected increase. Conidia were primarily released during the first 2 h of rainfall events. Inoculum production and release information were integrated into a dynamic disease-forecasting model for entomosporium leaf and berry spot (ELBS) disease on saskatoon.

The dynamic disease-forecasting model uses a disease pressure index (DPI) equation, which integrates information regarding the phenological development of saskatoon and the relationship of disease to inoculum potential and production, leaf wetness duration and temperature, inoculum release, host susceptibility to provide an estimate of disease pressure in an orchard. When the model was evaluated in two Manitoban orchards over 3 years, a strong correlation between predicted disease pressure and observed disease symptom development was recorded. After the field data were combined, the model was able to account for 82% of the variation in the increase of average lesion number per leaf observed during the pre-harvest period. Once control thresholds together with application and control guidelines for Topas 250E were added to the model, it was evaluated in a field trial at the Carman 2002 site. Based on only two spray recommendations from the model, good disease control was realized. Future work is required to establish economic thresholds for control when validating the model on different cultivars and geographic regions.

3. TECHNICAL REPORT

3.1. Background

Entomosporium leaf and berry spot develops rapidly under appropriate environmental conditions and damages fruit, making them unmarketable. This disease can virtually eliminate the production of a marketable crop in a particular year. Although there are fungicides registered for use against *Entomosporium*, their timely application is necessary to maximize control. The ability to predict conditions that may promote the disease well in advance has the potential to increase the chance of effective control, and also may serve as the basis for the development of alternative cultural methods to help control the disease. There are now excellent tools available for developing models to predict stages of plant development and the outbreaks of pests. These tools could be used as a basis for developing disease-forecasting models for saskatoons, and in addition, the information could be used to build phenological models of plant growth and fruit development in this crop.

The assessment of the phenological stages of development in the saskatoon in relation to meteorological factors also would be a valuable tool for efficient and successful crop management and marketing. Little is known about how vegetative development of the saskatoon is affected by environmental conditions such as weather. This information would be valuable for predicting orchard growth and development. Accurate prediction of developmental growth stages or phenophases of saskatoons would be useful for the proper timing of orchard operations, such as fruit thinning, irrigation, pest management, cultivation and harvest, which is based on the development of plant parts rather than calendar days. Maturity prediction models have been developed for several fruit crops. These include apple, apricot, prune and peach. Past and present weather conditions could serve as an environmental input for a predictive model of plant development. It would be beneficial to producers to be able to predict harvest dates. The correct timing of fruit harvest is important to maximizing marketable yield and fruit quality.

An accurate prediction of saskatoon growth stages would be useful for timing orchard operations, such as pest control and harvest. For example, the timing of fungicide applications could be based on saskatoon plant development and its relative susceptibility to disease. Ronald et al. (2001) showed that as saskatoon leaves mature they become less susceptible to ELBS disease. Therefore, knowledge of the physiological development of vegetative tissue could be important for determining the need for fungicide applications. The correct timing of saskatoon fruit harvest is also important for maximizing marketable yield and fruit quality (St-Pierre 1997). If past weather conditions could be used to predict the time of maximum marketable yield in advance, it would provide growers with time to coordinate harvesting efforts. Inability to plan and conduct harvest operations at the optimum time can lead to a harvest of overripe fruit. Harvested fruit that are overripe does not store well and can make processing difficult.

The limiting factor most affecting the quality and quantity of saskatoon fruit yield is ELBS disease, which is caused by the fungal pathogen *Entomosporium mespili* (D.C.) Sacc. In its asexual state *E. mespili* causes necrotic spots on leaves, fruits, and succulent stems of more than 60 species of plants, all in the subfamily *Pomoideae* of the rose family (Davidson 1989; Sinclair et al. 1987). The disease damages fruit, making them unmarketable, and reduces plant vigour, ultimately lowering yields. Entomosporium leaf and berry spot disease can eliminate the entire marketable yield of a saskatoon crop in any particular year.

As a problem on cultivated saskatoon plants, ELBS disease was first identified in nursery beds at the PFRA Shelterbelt Centre, Indian Head, Saskatchewan, in 1980, where it caused severe defoliation (Davidson 1990). In 1981, Davidson conducted an informal survey of orchards and wild stands from Ft. Vermilion, Alberta to Edmonton, and found the leaf spot and blight phases of the disease at all locations. The disease seemed to subside and was of minor importance until 1990, when it again caused havoc in central and north central areas of Alberta, resulting in large economic losses to growers (Davidson et al. 1991). By 1994, the prevalence of *E. mespili* in parts of Alberta had increased to the extent that most of the saskatoon fruit crop that year was destroyed (Lange and Bains 1994). The majority of orchards lost 80 to 90% of their crop, and cumulative financial losses were estimated at $1,000,000 CAD. Several years later, in 1999, the disease caused serious financial losses in Saskatchewan and Manitoba (Bains 2000). Entomosporium leaf and berry spot may even kill young saskatoon plants if the disease severity becomes sufficiently high.

The overall goal of this research project is to develop a model based on weather variables that 1) predicts the timing of important plant development stages such as flowering and fruit harvest, and 2) integrates plant growth and development with pathogen epidemiology to forecast ELBS disease severity on saskatoon. To accomplish this goal the main objectives are: 1) to develop a phenological model for forecasting growth and development of saskatoon; 2) to determine the leaf wetness duration and temperature required for infection of saskatoon leaves by *E. mespili* conidia under controlled conditions; 3) to estimate inoculum production relative to infected leaf area and examine environmental conditions required for stimulating release of *E. mespili* conidia; and 4) to design and test a dynamic disease-forecasting model for assessing and controlling ELBS disease pressure on saskatoons through the incorporation of information from objectives 1 to 3. The processes, assumptions, and ultimate construction of these models will be discussed in detail.

3.1.1. Important Phenological Stages for Predicting Saskatoon Fruit Maturity

Phenology can be defined as a branch of science that is focused on the study of a plant's biological development relative to climatic conditions (Dethier 1978). When studying phenological development, a clear definition of each phenological stage must first be determined. For example, the important phenological stages leading up to fruit set on the saskatoon plant have been previously defined. Friesen (1986) created a phenological growth scale to model plant development; St-Pierre (1997) created a simplified but comprehensive phenological scale for orchard management; and Sumner et al. (1999) created a phenological scale to study pollen development relative to the phenological stages of inflorescence expansion.

In St-Pierre's (1997) system, floral bud expansion was divided into 9 stages: dormancy, silver tip or bud break, green tip, white tip, tube, balloon, full bloom, petal fall and fruit set. A bud was considered dormant until it begins to swell in the spring ultimately resulting in bud break. As the dormant bud swells, the outer scales separate and pubescent hairs of the inner scales are revealed, resulting in a silvery appearance. After the bud breaks it continues to expand revealing a green colored inflorescence structure. Later, in the green tip stage, much of the inflorescence is exposed and the floral bracts subtending individual floral buds become visible. Between the green and white tip stages the first foliage leaves begin to expand near the base of the inflorescence, the entire developing inflorescence emerges from the bud, and the white petal tips of each individual flower become visible. During the tube stage, the tips of each individual flower continue to emerge but remain tightly closed. The tube phase is relatively short, and once the petals begin to open the flowers are considered to have reached the balloon stage of development. Once the petals have completely unfolded to expose the reproductive structures of the flower, the inflorescence as a whole is said to have reached full bloom. After full bloom petals begin to abscise, and if the flowers were pollinated, fruit are set. Fruit set is characterized by a swelling of the flower's receptacle.

Steeves and Steeves (1990) defined anthesis (flowering) as petal spread and the start of petal drop as petal abscission. St-Pierre and Steeves (1990) used peak anthesis (the time at which greater than 50% of the flowers in an inflorescence were open) as a key reference point in describing phenology of the saskatoon. They also indicated that, for each year, saskatoon plants of the same genotype and cultivar reach anthesis at approximately the same time in each orchard and that the date of peak anthesis can vary between years over a two and a half week period in mid-to late-May. Variability in the time of peak anthesis is not unusual, as it can be caused by a variety of environmental factors. Tydeman (1964) observed that the full bloom of the apple cultivar 'Cox's Orange Pippin', varied from the 15th April to the 23rd May over a 43-year period, depending on temperature.

Fruit development and ripening also go through identifiable stages. An understanding of the physiological criteria that are used to determine when fruit are ripe must be understood in order to accurately identify the correct timing of harvest. Janick (1986) stated the proper harvest date for different pear cultivars is determined by a combination of criteria, among which are pressure testing of the fruit, ground color of the skin, seed color, and percentage of soluble solids in the flesh. In some crops, other criteria include the formation of abscission zones separating fruit from the stem (muskmelons), the visible stage of development (such as the degree of bud tightness in roses), color (tomatoes), sugar-acid balance (blueberries), and even the way fruit sounds when thumped (watermelons).

Saskatoon fruit are characterized as pentalocular pomes (McGarry et al. 1998; St-Pierre 1997). Rogiers and Knowles (1997) developed a 9-color maturity class index for saskatoon fruit based on chemical and physical characterization. It was proposed that this scale be adopted for future studies on saskatoon fruit growth and development. Although variability was quite considerable between the different saskatoon cultivars, basic trends in chemical and physical maturation were observed. Chemical changes during maturation included a decrease in pH and organic acids, and an increase in soluble solids (Rogiers and Knowles 1997). Changes in respiration and ethylene production were also characterized during maturation and ripening of saskatoon fruit (Rogiers et al. 1998). Physically, as a saskatoon fruit ripens, its diameter and fresh/dry biomass increases while its firmness decreases. In relation to overall fruit size, mature saskatoon pedicel and fruit size increase linearly with the number of developed seeds, suggesting that saskatoon seeds influence growth of the surrounding tissues (McGarry et al. 1998). Gorchov (1985) showed that fruit growth is correlated with the number of developed seeds per fruit in *Amelanchier arborea*. The color of the fruit changes from a green to light red to deep purple as the fruit ripen. The color changes associated with maturation and ripening of saskatoon fruit involves both chlorophyll degradation and anthocyanin biosynthesis (Rogiers and Knowles 1997). Above all, it seems the color changes associated with fruit ripening are the most important factor for determining harvest date. Although the maximum fruit weight is not yet realized until stage 9, St-Pierre (pers. comm.) suggests that harvesting fruit which are overripe ($\geq$ stage 9) could reduce the storability and processing desirability of the fruit. Therefore, according to the scale developed by Rogiers and Knowles (1997), saskatoon fruit harvest should begin when the fruit reach the 8th stage of development. Fruit that have reached stage 8 are described as being dark red with purple hues. Saskatoon fruit are considered climacteric in nature (Rogiers and Knowles 1997), and therefore should continue to ripen as ethylene levels increase after harvest has occurred. However, the ripening of harvested saskatoon fruit can be inhibited if the ethylene producing precursor 1-aminocyclopropane-1-carboxylic acid (ACC) is missing (Rogiers et al. 1998). The synthesis of ACC is likely the rate-limiting step of ethylene production in harvested saskatoon fruit.

3.1.2. Modeling Phenology – Model Construction and Assessment

The history of phenological modeling goes back at least as far as the 1700s. Reaumur (1735) first suggested that the time required for plants to complete a phase of development could be more accurately estimated from temperature sums than from calendar days. Entomologists were the first to develop and use phenological forecasting models for predicting stages of insect life cycle development. This is because of the significant impact insects have on important agronomic crops. Numerous studies have produced significant evidence that feeding by insect herbivores can influence the growth, reproduction, and population density of native herbaceous plants (Crawley 1983; Parker 1985; Hendrix 1988; Weis and Berenbaum 1989; Louda 1989, 1995). Insects are organisms that cannot internally regulate their own temperature, and therefore respond to environmental temperatures to regulate their growth and development. Insects require a certain amount of heat in the form of ambient temperature to develop from one phenological stage to the next. For example, AliNiazee (1979) used heat unit accumulation for predicting important developmental events of fruit fly (*Rhagoletis indifferens*) on cherry trees. Gangavalli and AliNiazee (1985) used the same heat unit accumulation approach to model development of the obliquebanded leafroller, *Choristoneura rosaceana* (Harris). Black cutworm, alfalfa weevils, and stalk borer moths are just a few more insect examples for which the amount of ambient heat required to reach maturity is known (http://www.ipm.iastate.edu/ipm/icm/1998/4-6-1998/dd.html, last accessed on March 8, 2003).

Much like insects, plants do not generate their own heat, and thus require and respond to heat from the environment to grow and develop. Dethier (1978) observed a direct relationship between the phenological development of a plant and past and present weather conditions. Physiological time provides a universal reference for the development of an organism based on the accumulation of temperature-based heat units (Strickler 1998). The temperature experienced by the plant may be thought of as an empirical analog for the amount of physiological time that the plant has experienced (Hodges 1991a). In essence, because temperature drives most components of plant development, it can be used to predict the occurrence of plant stages based on the accumulation of heat units (Eggert 1960; Richardson et al. 1975; Smith 1985; van den Brink 1974). Various linear (Cate and Hsu 1978; Yan and Wallace 1998) and nonlinear (Brown 1969; Kiniry and Keener 1982; Major et al. 1975; Wassink 1974) algorithms have been developed to calculate physiological time, which is then used to predict plant development. Given that the saskatoon is a temperate woody plant producing preformed floral and vegetative organs (Steeves and Steeves 1990), the easiest way to monitor its phenological development is to measure the number of heat units required to reach each stage of plant development during the growth season after organ initiation (Anstey 1966). This same method was used to monitor the physiological time required for plants like apple, pear, cherry, peach and apricot, to reach flowering (Anstey 1966; Sisler and Overholser 1943). Junttila et al. (1983) has shown that the dehardening of saskatoon reproductive buds is dependent upon a relatively unvarying sum of heat units.

Temperature is known to influence saskatoon dormancy, bud break, and even governs plant survival. For example, a population of 'Smoky' saskatoon plants requires at least 1500 to 2100 h at temperatures between 0 and 7°C in order to fulfill their chilling requirement and have uniform bud break (St-Pierre, pers. comm.). Bud break, or the onset of foliation of many perennial plants, can be predicted with spring warming models (Aber et al. 1996; Goulden et al. 1996; McMurtie et al. 1994). In Saskatoon, Saskatchewan, it was suggested that the optimum temperature for saskatoon bud break could be anywhere between 15 and 25°C and could also be cultivar dependant (Kaurin et al. 1984). Bud break, also defined as rest completion for perennial plants, used as a physiological parameter for the starting point of heat unit accumulation, has been found to be superior to simply using a fixed calendar date (Eiesensmith et al. 1980; Richardson et al. 1974). Overwintering flower buds of saskatoons are very hardy, with dehardening proceeding slowly under natural conditions (Junttila et al. 1983). Olson and Steeves (1983) showed a strong decrease in floral tissue hardiness at anthesis resulting in an increased vulnerability to frost damage. Symptoms of frost injury can vary among species and genotypes, various tissues, and with the ambient temperature and duration of exposure (Kozlowski and Pallardy 1997).

The flowering date of woody plants can vary due to differences in geographical region and temperature. Geographical locations can cause variations in the timing of phenological development (Steeves and Steeves 1990). For example, latitude and altitude can indirectly affect a plant's phenological development by influencing the amount of heat units which are accumulated in that growing region (Kozlowski et al. 1991b). Temperature influences flowering date because it affects aspects of shoot growth, including bud development, release of bud dormancy, and expansion of leaves, internodes, and inflorescences (Kozlowski and Pallardy, 1997).

Smith (1985) tested the influence of environmental variables like temperature, rain, and sunshine prior to the bloom of peaches and found post-bloom temperatures to be the only variable that had any significant effect on the development of fruit. Most phenological models for predicting harvest dates have been based on the sum of heat units during the post-bloom period (Smith 1985; Tukey 1942). With regards to model accuracy, Eggert (1960), using regression analysis, found that 92% of the variability in the total elapsed days from bloom to harvest could be accounted for when the date of apple maturity is predicted 40 d after petal fall.

Fruit growth and the timing of premature fruit abscission in saskatoons is similar to that of other angiosperm species in which fruit growth approximates a single sigmoid type of growth curve, and fruit abscission occurs prior to the period of exponential growth (Bollard 1970; McGarry et al. 1998; Stephanson 1980). The first phase of fruit biomass increase is characterized by an initially slow and then increasing rate of fruit development. The rate of growth is relatively constant in the second phase, and it is followed by a third period of logarithmic growth. Pome fruit such as apples and pears typically exhibit sigmoid growth curves with a single period of exponential fruit growth (Westwood 1993).

The earliest and most commonly used model for calculating heat unit accumulation is the growing degree-day (GDD) model, which calculates the daily accumulation of heat units from the mean daily temperature above a selected base temperature (Wolf et al. 1986). One day is the time unit and each degree above the base temperature is assumed to have a linear effect on plant growth and on the accumulation of heat units. The GDD equation used by Friesen (1986) to predict maturation of saskatoon fruit is shown in equation [1] as follows:

$$\text{GDD} = [(T_{max} + T_{min})/2] - T_{base} \quad [1]$$

where GDD represents the daily accumulation of heat units, T_{max} is the daily maximum temperature; T_{min} is the daily minimum temperature; and T_{base} which is the base temperature. Selecting an appropriate base temperature often involves using several base temperatures for calculating degree-days and then observing which most accurately predicts the organism's development (Morris et al. 1980; Seem and Szkolnik 1978; Smith 1985). Junttila et al. (1983) determined that 5°C was the base, or minimum temperature required for saskatoon plant development.

The GDD heat unit system used to monitor phenological development has a number of practical problems and limitations. There are some poor assumptions built into the GDD system. The heat unit theory assumes that there is only one base temperature through the life of the plant, and that day and night temperatures are of equal importance to plant growth. Unfortunately, these assumptions are not correct (Shaykewich 1995). Furthermore, overestimations can be made if the temperature rises above the optimum temperature. This is because the GDD system assumes a linear relationship between development and temperature with no maximum. This does not correspond to what actually occurs in nature (Morris et al. 1980), and therefore some modifications are necessary to improve model accuracy.

One way to account for the inaccurate linear response to temperature and overestimation of heat units in the GDD model is to use the corn heat unit (CHU) system for calculating heat unit accumulation. The CHU system recognizes the nonlinear nature of the growth response to temperature by incorporating an optimum temperature for plant development (Brown 1969). Originally, the CHU system was the most commonly used heat unit system for corn in Canada. Nevertheless, it still did not accurately reflect the response of the corn plant to temperatures in different growing regions. Attempts were made to improve the accuracy of the CHU equation by altering the base temperature (Cutforth and Shakewich 1989). However, the accuracy did not improve until minimum, optimum, and maximum temperatures were incorporated into the phenological model. From this concept the CERES-Maize model emerged (Kiniry 1991). It assumes that rate of development in the various stages is zero at temperatures $\leq$ 8°C, increases linearly from 8-34°C, then decreases linearly to zero at 44°C. Tests of this model revealed that the average error in estimating the time from planting to silking is 2 to 3 d (Kiniry 1991). Much like the CERES-Maize model, the physiological day (P-Day) model was developed for predicting various potato stages based on a minimum, optimum, and maximum temperature (Sands et al. 1979). This type of model assumes that the rate of plant development is zero at temperatures less than the minimum temperature, increases linearly between the minimum and optimum temperatures, then decreases linearly and becomes zero once the maximum temperature is reached. Daily accumulations of P-Day heat units are calculated from equation [2] as follows:

[2] $$\text{P-Day} = (1/24) \text{ X } [(5 \text{ X } P(T_1) + 8 \text{ X } P(T_2) + 8 \text{ X } P(T_3) + 3 \text{ X } P(T_4)]$$

where P-Day represents the daily accumulation of heat units, T_1 is equal to the daily minimum temperature (T_{min}); T_2 is derived from equation [3]:

[3] $$T_2 = ((2 \text{ X } T_{min}) + T_{max}) / 3$$

T_3 is derived from equation [4]:

[4] $$T_3 = (T_{min} + (2 \text{ X } T_{max})) / 3$$

and T_4 is equal to the daily maximum temperature (T_{max}). The denotation for P is a heat unit value that is calculated differently for different values of temperature (T): 1) equal to zero when the T is less than the minimum temperature required for plant development (7°C); 2) calculated according to equation [5] when T is greater or equal to 7°C and less than the optimum temperature required for plant development (21°C):

[5] $$P = k \text{ X } (1 - [(T - 21)^2 / (21 - 7)^2]$$

3) calculated according to in equation [6] when T is greater than or equal to 21°C and less than the maximum temperature required for plant development (30°C):

[6] $$P = k \text{ X } (1 - [(T - 21)^2 / (30 - 21)^2]$$

and 4) is equal to zero when T is greater than or equal to 30°C. The denotation for k represents a constant of 10. The sum of daily heat units produces an estimate of organism growth and development, relative to physiological time. To improve accuracy, it would be useful to modify the model to incorporate minimum, optimum, and maximum temperatures that affect saskatoon plant growth and development.

Regression and coefficient of determination analysis can be used to determine the most reliable time for a plant to reach a particular phenological stage based on the sum of heat units calculated over a number of years. Baker et al. (2001) and Eggert (1960) used this system to create phenological models for muskmelon and 'McIntosh' apple development, respectively. Both linear and quadratic regression equations provide an accurate prediction of the date of saskatoon fruit harvest based on the sum of GDD heat units after full bloom (Friesen 1986). The quadratic regression model accurately predicted harvest within ±1 day, when it was started 40 d after full bloom, for the four years in which they were developed. It should be noted that there is a difference in the GDD heat unit requirements among different cultivars of saskatoons. For instance, 'Smoky' and 'Pembina' saskatoon plants required significantly fewer heat units to reach full bloom than did the cultivar 'Thiessen'.

A model that successfully simulates a measured set of data does not necessarily indicate its predictive ability across a range of conditions. Model validation requires independently derived measurements that must be validated across a variety of microclimates over a number of years to ensure widespread applicability (http://www.ipm.ucdavis.edu/DISEASE/california_pestcast.html, last accessed on March 8, 2003). The accuracy of different models can be measured also by comparing the standard deviation of heat units required for a plant population to reach a particular phenological stage of development over a number of years (Gauch 1988). Predicted values of phenological development can be plotted against measured values to visually display the reliability of phenological models (Yan and Wallace 1998). For example, Baker et al. (2001) showed good model accuracy when predicted muskmelon node development was plotted against observed node development.

Accurately modeling plant phenological development from weather data allows for advanced planning and ultimately provides the grower with an economic advantage (Shaykewich 1995). Crop growth and yield are typically modeled in response to weather conditions in a given environment (Pace and MacKenzie 1987). Phenological modeling has been used to predict the date of full bloom and has provided growers with an advanced warning system for spray and thinning programs in apple, pear, cherry, peach, and apricot crops (Anstey 1966; Sisler and Overholser 1943). Wheat crop yield based on the season's growing conditions can also be predicted (Raddatz et al. 1994). Up to 69% of the observed variance in spring wheat yield could be explained by agrometeorological variables. Being able to predict yield gives economists the ability to produce economic forecasts for a region. For example, organizations like the Canadian Wheat Board use such information to plan transport, storage, and trade of wheat (Raddatz et al. 1994). To date no model has been developed to predict saskatoon fruit yield.

As discussed earlier, temperature is particularly important for governing a plant's phenological development (Eggert 1960; Richardson et al. 1975; Smith 1985; van den Brink 1974). However, the temperature directly affecting plant development is difficult to accurately monitor because plant growth and development actually depends on leaf rather than air temperature. Since leaf temperature data is not commonly available, air temperature data must be used to provide an estimate of leaf temperature. Both moisture stress (Ehrler et al. 1978) and solar radiation (Caprio 1974) can influence the temperature acting on a leaf surface and thus reduce the time required to reach a certain phenological stage by causing the temperature of the plant tissue to become greater than the ambient temperature.

Besides temperature there are several other known environmental variables that can influence a plant's phenological development and change morphological characteristics, including yield. Some of these environmental factors include drought, frost, defoliation, and lack of sufficient resources. Fraser (1962), Kramer (1962), and Zahner (1962) studied the effect of water deficits on both shoot extension and cambial activity and found that growth is limited when soil moisture levels are low. Frost can also do direct damage to yield by causing abscission of flowers and fruit (Addicott and Lyon 1973; Garwood and Horvitz 1985; Motten 1982). Defoliation (Bentley 1977; Janzen 1976; Kozlowski and Keller 1966; Rockwood 1973; Stephanson 1980; Willson and Price 1980) and lack of sufficient resources (Janzen 1971; Lloyd 1980; Quinlan and Preston 1968; Salisbury 1942; Stephanson 1980; Tamas et al. 1979; Udovic and Aker 1981; Wyatt 1980; Willson and Price 1977) have been shown to affect physiological development and cause flower and fruit abortion in many plant species. Fungal pathogens can also adversely affect the plant's health, and ultimately influence phenological development (McMullen et al. 1997). St-Pierre (1989) indicated that environmental factors like frost, insect, and fungal damage to vegetation and flowers could affect the phenological development and ultimately the yield of saskatoons. Although little is know about how the fungal pathogen *Entomosporium mespili* (D.C.) Sacc., which causes entomosporium leaf and berry spot disease, affects phenological development it can be the one of the most significant environmental factors limiting saskatoon fruit production (Lange et al. 1998).

3.1.3. Entomosporium Leaf and Berry Spot Disease

Entomosporium mespili is spread to susceptible host tissue primarily by means of its distinctive, asexually produced conidia. Conidia are formed from conidiogenous cells, which are found inside the acervuli (Mims et al. 2000). During its lifetime, three to four conidia can arise from each conidiogenous cell. Collectively, conidiogenous cells produce enormous numbers of conidia which are primarily responsible for the propagation and dissemination of the pathogen (Alexopoulos 1962). Conidia have been referred to as summer spores because they are produced only during the growing season in temperate regions. Conidia produced on living leaves of temperate *Amelanchier* species germinate readily from May to August (Hori and Kobayashi 1980a). In a laboratory study, Baudoin (1986a) was able to produce conidia on artificial media.

Leaves, fruit, and young branch tissue can all be infected by *E. mespili* conidia. Leaf lesions on pear caused by *E. mespili* conidia arise as minute dots and when fully developed they appear as brown to gray irregular shaped spots, 2-5 mm in diameter (Rosenberger 1981; van der Zwet and Stroo 1985; Sinclair et al. 1987). Infected leaves of *Amelanchier* species turn yellowish or reddish with a greenish halo around the necrotic spots (Hori and Kobayashi 1979) (see Appendix C Fig. C.2). Entomosporium leaf and berry spot lesions may be become so numerous that they coalesce to form large necrotic blotches that cover much of the leaf surface (Sinclair et al. 1987). Eventually premature leaf abscission may result if the infected area becomes too great. Lelong (1889) showed that pear fruit production is adversely affected by pathogens that defoliate the plant. Saskatoon fruit quality and quantity can suffer when photosynthetic area is lost to spotting and untimely defoliation of the plants in late spring and early autumn (Hori and Kobayashi 1979; St-Pierre 1997). Not only will susceptible host plants like saskatoons become defoliated, their fruit can also be spotted, deformed, and cracked, reducing marketable yield significantly (Sinclair et al. 1987). Symptoms on saskatoon fruit include premature ripening of fruit and watery gray lesions that expand and eventually coalesce. St-Pierre (1997) states that if more than 6% of the saskatoon fruit surface area becomes infected, the fruit are considered unusable even for processing. Much like saskatoons, ELBS symptoms on other genera like *Pyrus* begin as pinpoint spots on fruit but coalesce to form large necrotic blotches (McAlpine 1911). In addition to leaf and fruit infections, young branch tissue can also be infected by *E. mespili* conidia. Only young, 1-year-old branch tissue of pear is susceptible to infection by *E. mespili* (van der Zwet and Stroo 1985). Branch lesions caused by leaf blight of pear will begin as small, circular spots but later increase in length and become sunken (Stewart 1915). If a lesion girdles the stem, the portion above the diseased area can die (Lelong 1889). Highly susceptible species like the saskatoon can have twig and branch dieback if they are defoliated in several successive years (Sinclair et al. 1987).

Conidia can overwinter on young branch tissue, dead infected leaves that remain on or around the plant, and possibly on infected pedicels and fruit. Hori and Kobayashi

(1980a) showed that *E. mespili* conidia could overwinter within the cankers formed on young green twigs of *Crataegus oxyacantha* Lindm. Conidia released from infected branch tissue seem to be the primary source of inoculum for the next year's new leaves (Goldsworthy and Smith 1938; Kudo and Takanashi 1976; and van der Zwet and Stroo 1985). Aside from branch cankers, conidia of *E. mespili* can also be produced on detached and dead leaves that overwinter on or around the plant (Stathis and Plakidas 1959). However, Bains (1999) found that when leaf litter and infected fruit were removed from saskatoon plants in the fall, the disease was still able to flourish when provided with ideal environmental conditions in the spring. It has also been suggested that infected saskatoon fruit and pedicels that remain attached to plants might provide inoculum for initiation of the disease in subsequent years (Bains 1999).

Once new leaves begin to expand from the buds, they become exposed to infection by *E. mespili* conidia. Water, animal, and insect vectors are required to move conidia to new host tissue (see also section 2.2.4.2). Ronald and St-Pierre (2002) showed that conidia become available for dissemination when the upper surface of the acervulus breaks. According to Funk (1985), rainwater then physically moves the conidia from overwintering acervuli to the young expanding leaves. St-Pierre (pers. comm.) suggested that *E. mespili* conidia could be moved over great distances by vectors such as birds and insects. For instance, when a vector organism lands on an infected plant during a wet period, water-containing conidia may adhere to its body, and when it moves to another plant these conidial spores would be carried with it. Ascospores may also play a role in the dissemination of ELBS over great distances; however, no research to date has examined this phenomenon in saskatoon orchards.

Entomsporium mespili can be spread rapidly because of the pathogen's short incubation period and polycyclic nature. The incubation period is defined as the time period between initial infection and disease symptom development. The pathogen, which causes ELBS disease, produces conidia rapidly yielding many new generations in a single season (Sinclair et al. 1987). When *E. maculatum* conidia are disseminated onto a susceptible host like *Pyrus*, they can germinate within 18 h and then form appressoria which penetrate the leaf cuticle in less than 48 h (van der Zwet and Stroo 1985). One to two germ tubes usually germinate from each conidium, although each of the cells that comprise a conidium is capable of germinating (Stowell and Backus 1966). The lateral cell bodies of the conidium germinate less frequently than the apical and basal cells (Hori and Kobayashi 1979). Subsequent infection within a leaf occurs when hyphae spread subcuticularly, later invading host cells with haustoria (van der Zwet and Stroo 1985). Haustoria are like appressoria in function, except they are exclusively used to penetrate neighboring host cells. Leaf and fruit lesions appear on pear 4 to 7 d after inoculation (van der Zwet and Stroo 1985). Acervuli form as dark, blister-like, irregular shaped structures beneath the cuticle in the center of lesions (Rosenberger 1981). The size of acervuli varies with host and environment, and can be more than 1 mm in diameter (Sinclair et al. 1987). Acervuli caused by *E. mespili* appear as either single, or multiple, confluent raised areas on the adaxial and abaxial surfaces of infected *Photinia* and saskatoon leaves (Mims et al. 2000; Ronald and St-Pierre 2002). The production of conidia puts upward pressure on the cuticle of saskatoon leaves causing it to rupture

(Ronald and St-Pierre 2002). White masses of conidia can be observed exuding from mature acervuli 2 to 4 wk after initial infection on *Pyrus* (van der Zwet and Stroo 1985). Mycelia growing in plant tissue will continue to produce acervuli as long as environmental conditions are favorable (Alexopoulos 1962). The formation of conidia begins on diseased material early in the summer in temperate areas, but can continue all year in regions that have a mild winter (Sinclair et al. 1987).

In autumn, *E. mespili* can shift from anamorphic conidial production to a teleomorphic state (Sinclair et al. 1987). Greene (1942) was the first to observe ascocarp initials, which began to appear in the last half of September and continue to form until November on *Crataegus*. After the infected leaves fall from the plants, it was said that the apothecia break through the epidermis and complete their development in the spring. Apothecia, which look like abnormally small acervuli appear primarily on the lower surface of *Crataegus* leaves and eject colorless, two-celled ascospores into the air (Stowell and Backus 1967). As in many other Ascomycetes, asci at different stages of development are present in any given ascocarp, with the result that the release of ascospores may continue for a considerable period of time. It was also shown that conidia could form in place of the asci in the apothecia. The role of ascospores in initiating disease in spring is unknown, but is generally considered to be unimportant (Sinclair et al. 1987). Horie and Kobayashi (1980a) observed no formation of the perfect stage on overwintering diseased leaves of several host species, which included *Crateagus*. Currently, there is no evidence to suggest that *E. mespili* completes a full sexual reproduction cycle on *Photinia* (Mims et al. 2000), or *Amelanchier* plants.

3.1.4 Environmental Factors that Influence Pathogen Epidemiology

Disease results from the interaction of a pathogen with a susceptible host in a favorable environment over time (Fry 1982a). All three of these components, which make up the disease triangle, must be present in order for disease to develop (Agrios 1997). For example, *E. mespili* cannot infect woody plants outside of the rose subfamily *Pomoideae*, despite having ideal environmental conditions and copious amounts of inoculum present. If a pathogen like *E. mespili* is present in an ecosystem with susceptible hosts, climatic factors will ultimately influence the success of ELBS disease development.

The weather conditions acting on a pathogen are of particular importance for directing disease development. However, the use of weather as a primary input for studying disease development has lead to some application problems (Schroedter 1983). Historically, there are two problems with this type of system. The first problem being that typical meteorological measurements have been taken at a standard height out in an open field. This type of measurement does not accurately represent the dynamic environmental conditions within the crop canopy (Harrison 1992). A second and more serious problem arises from the fact that a network of meteorological stations is in most cases a wide-meshed one, incapable of taking into account all the regional differentiations and local characteristics which are important for studying epidemiological processes.

New real-time weather monitoring networks that can be set up to intensively blanket-growing regions have made it possible to assess the importance of environmental factors on disease development. The Adcon Telemetry and Precision Vantage systems are examples of networks that have helped improve environmental variable monitoring by providing users with a weather monitoring system that can take measurements from within the plant canopy at frequent intervals (real-time) (Hart 2001). For example, the Adcon Telemetry system will take environmental measurements like temperature, precipitation, and leaf wetness and transmit the data from within the plant canopy to a server computer, process it, and ultimately suggest control measures to end users based on program models (Clarke 1996; Oltman 1996; Rieger 1995). In order to utilize this valuable technology in the management of ELBS disease on saskatoon plants, a disease-forecasting model incorporating environmental factors that influence disease development must first be developed.

Temperature will affect the physiology of each plant pathogen differently. It is clear that some specific minimum, optimum and maximum temperatures are necessary to drive each pathogen's growth and development. Of all the environmental factors, temperature is the easiest to measure and control so its effects have been investigated extensively for many crops. Tamm and Flückiger (1993) observed sweet cherry infection by *Monilinia laxa* (Aderh. & Ruhl.) Honey, and found mycelia would grow between 2.5°C and 30°C, with an optimum at 24°C. In addition, they found that the maximum germination fraction of conidia reached 98% when suspended in deionized water and exposed to temperatures between 15°C and 25°C. Thompson and Jenkins (1985) studied cucumber infection by *Colletotrichum lagenarium* (Pass.), and showed that conidial production commenced when lesions became visible and was greatest at 24°C. They also showed that lesion size increased as the temperature was raised from 20°C to 32°C. The optimum temperature for infection of *Photinia* leaves by *E. mespili* was about 20°C, and infection was only slightly retarded at 15°C and 25°C (Baudoin 1986a). To date there has been no study examining the effect of various temperatures on the development of ELBS on saskatoon plants.

In addition to the necessity of having ideal temperatures for disease development, leaf wetness is also a prerequisite for fungal infections. For example, precipitation, dew formation, drizzle, fog, and mist all result in wet foliage, which is a prerequisite for potato infections by *Phytophthora infestans* (Mont.) de Bary. In a review paper, Harrison (1992) indicated how environmental moisture is important for the germination of, and infection by, *P. infestans* sporangia and zoospores, growth of hyphae, production of sporangia, dispersal of sporangia, survival of spores and hyphae, susceptibility of plant tissue and ultimately development of an epidemic. Hong et al. (1998) showed that there is a negative logarithmic relation between *Metarhizium flavoviride* Gams & Rozsypal conidium viability and moisture content. Green and Bailey (2000) reported that 8 hr of leaf wetness are required by *Alternaria cirsinoxia* conidia to germinate and induce infection on Canada thistle. Baudoin (1986a) showed that 9 to 12 h of leaf wetness is a pre-requisite for substantial infection of *E. mespili* on *Photinia*, and as little as 5 to 6 h can sometimes produce a few leaf spots at an optimum temperature. The number of leaf wetness hours required to induce *E. mespili* infection on saskatoon plants is unknown.

Precipitation not only influences sporulation and infection success, it can also be required to disperse the reproductive structures of those fungi which do not rely on wind for their distribution. In potatoes, splash droplets containing spores, produced when raindrops hit sporangia on a leaf or stem, are responsible for transporting *P. infestans* inoculum over short distances within a crop (MacKenzie et al. 1983). Madden et al. (1996) showed that as rain intensity increases so does the splash dispersal of *Colletotrichum acutatum* Simm. conidia, and as a result, disease levels can also be heightened. Jenkinson and Parry (1994) showed that raindrops 5 mm in diameter will cause *Fusarium culmorum* (W. G. Smith) and *Fusarium avenaceum* (Fr.) Sac. conidia to be splashed vertically (mostly within 20 cm) and horizontally (mostly within 30 cm). Pinkerton et al. (1998) showed a quantitative relationship between the hourly capture of *Anisogramma anomala* (Peck) E. Müller ascospores and the number of hours since the beginning of a precipitation event. They found that as the duration of the precipitation event increases, so does the number of ascospores released up to 12 h, and then after that time the numbers begin to decrease. Francl (1997) showed that prolonged wetness periods were required for the release of *Pyrenophora tritici-repentis* (Died.) Drechs. conidia in wheat fields. Similarly, Rosenberger (1981) observed dissociation of *E. maculatum* conidia from acervuli on pear leaves 1 h after being subjected to free water; however, the effect of precipitation on *E. mespili* conidium dispersal has not been quantified from infected saskatoon leaves.

When moisture evaporates from a plant's surface the relative humidity (RH) increases just above that surface. This increase in RH in turn favours disease infection and development. Lowering RH (50 to 80%) in a storage environment can significantly decrease the severity of *Botrytis cinerea* Pers.:Fr. on cut roses (Hammer and Marios 1989). Utrata (1980) showed that RH is essential for the development of potato blight in the field. A RH of 64 to 72% is required for the development of powdery mildew of mango between the temperature range of 11 to 31°C (Gupta 1989). Platford (pers. comm.) suggested that periods with RH greater than 90% could be just as significant for causing sclerotinia to develop on canola as the same period of leaf wetness.

The effect of RH on the development of an epidemic is difficult to quantify accurately. This is because RH can vary in time and position relative to the soil surface, as it continuously interacts with changing environmental factors (Harrison 1992). Rose (1966) and Monteith (1973) demonstrated that ambient humidity, air speed, and plant architecture are interdependent in determining the RH of air at a point just above the surface of a leaf or stem.

Ideal combinations of temperature and moisture in the form of precipitation and (or) RH are required for a fungal pathogen to infect a host plant. The effects of temperature and leaf wetness duration on conidium germination and infection have been studied for numerous crop/pathogen interactions. More *Venturia inaequalis* (Cooke) G. Winter ascospores are released from apple leaves when the perithecia have been warmed or subjected to a humid atmosphere before they are wetted (Brook 1969a). Gilles et al. (2001) showed how the infection, incubation period, and asexual sporulation of

Pyrenopeziza brassicae Sutton & Rawl are important components of disease progress on oilseed debris, and how all are affected by a complex interaction between temperatures that range between 5 and 18°C and length of the leaf wetness period. Mathieu and Kushalappa (1993) showed that increasing periods of leaf wetness and an optimal temperature of 25°C promoted celery infection by *Septoria apiicola* Speg. When more than 2 h of leaf wetness, and optimal temperatures between 21 and 24°C were provided, *Colletotrichum orbiculare* (Berk. & Mont.) Arx inoculum infected watermelon plants (Monroe et al. 1997). Maximum germination of conidia of *Botryosphaeria obtusa* (Schwein) occurred in free water and declined as RH was reduced from 100 to 92%; no germination was detected at 88.5% RH (Arauz and Sutton 1989). Pfender (2003) used wetness degree-hours, which is a function of moisture duration and temperature, to successfully model the infection success of *Puccinia graminis* subsp. *graminicola* Z. Urb. on perennial ryegrass seed crops. Degree-hours are calculated by multiplying the hours of leaf wetness by the average temperature above a lower threshold during that period. For example, 3 h of leaf wetness at an average temperature of 5°C equals 15 degree-hours.

Temperature and leaf wetness are important environmental factors that drive ELBS disease symptom development on susceptible host plants. If the leaf surface is wet for an extended period of time (> 6 h), the optimum temperature for infection of *Photinia* leaves by *E. mespili* was around 20°C (Baudoin 1986a). Furthermore, the minimum and maximum temperatures at which this pathogen could infect *Photinia* were roughly 10°C and 30°C respectively. Results from Baudoin (1986a) support those of Rosenberger (1981) on ELBS of pear for an optimum temperature between 20°C and 25°C and the shortest wetness period for infection as 8 h. In vitro, conidia of *E. mespili* which were isolated from an *Amelanchier* host plant germinated between 5°C and 30°C with an optimum at 26°C (Hori and Kobayashi 1979), and subsequent growth of the germ tubes occurred between 22°C and 26°C. Next to no infection by *E. mespili* has been reported beyond 30°C (Hori and Kobayashi 1979; Yamaguchi 1977). Yamaguchi (1977) tested the effect of temperature on mycelial growth of an *E. mespili* isolate originating from *A. asiatica*. The isolate grew best at 20°C and did not grow above 30°C. Davidson et al. (1991) stated that ELBS disease symptom development in saskatoon orchards is highly dependent on the weather, requiring both moisture and warmth for optimal growth. Disease surveys performed in Alberta during 1993 and 1994 further supported this interpretation (Lange and Bains 1994, 1995). Knowledge of what combinations of leaf wetness duration and temperature promote ELBS disease symptom development on saskatoon plants would be useful for creating a forecasting model to aid in controlling the disease.

In addition to temperature, leaf wetness, and RH, other environmental factors, such as solar radiation and wind, can influence the success of pathogen infection. Radiation emitted by the sun can adversely affect disease development. For example, De Weille (1963, 1964) showed that the viability of *P. infestans* sporangia falls from 65% to 40% when they are exposed to diffuse sunlight for 2 h. Solar radiation reduces spore viability by warming the leaf surface and surrounding air temperature. The increase in temperature would consequently lower the RH surrounding the leaf. This increase in temperature and decrease in RH would have a drying effect on the spores and could ultimately decrease their vitality and infection success. After *E. mespili* conidia are deposited onto *Photinia* leaves, they can survive dry periods for up to 12 h at 25°C in darkness or in the shade with little loss of viability, regardless of whether the dry period occurred immediately after inoculation or after 5 h or 12 h of leaf wetness (Baudoin 1986a). Only bright sun and high temperature resulted in a significant loss of infectivity. Wind will also affect spore survival. For instance, a strong wind blowing through a potato crop may dry sporangia that are still attached to sporangiophores and lead to decreased viability (van Everdingen 1935). Wind affects other environmental parameters by mixing the air within a crop canopy. Mixing of the air moderates differences in temperature and RH in a vertical plane from soil level to above the top of the canopy, and decreases the differences in the horizontal plane so that the environment around the plant becomes drier as wind penetrates the crop (Harrison 1992).

3.1.5 Control of Entomosporium Leaf and Berry Spot Disease

Fungicide applications are an important tool that can be used to control fungal epidemics on valuable horticultural and ornamental crops. Foliar sprays of potato plants with phosphonic acid substantially reduce the infection of tubers by *P. infestans* (Cooke and Little 2002). Ergosterol-biosynthesis inhibiting and chlorothalonil fungicides were found to be effective for controlling entomosporium leaf spot on *Photinia* (Bowen et al. 1994). Even with fungicide application, Lambe and Baudoin (1984) indicate that ELBS disease is one of the most difficult diseases to control on *Photinia*. They noted that to avoid leaf spotting and early season defoliation, fungicide applications must begin when spring foliage is rapidly expanding and continue until growth is complete. It was clear that fungicides must be applied at the beginning of the season to protect plants from early infection. Because *E. mespili* is polycyclic, infections caused early in the season will increase potential inoculum levels, which will in turn enhance the possibility of significant fruit infection later in the growing season (Bains 2000).

There are three fungicides registered to control ELBS disease on saskatoons in Canada: Funginex (triforine), Topas 250E (propiconazole), and Kumulus (sulfur) (Bains 2000). These three fungicides can be separated into two groups, systemic and contact. Funginex and Topas 250E are systemic fungicides, meaning that they can move into and throughout the plant body, and in doing so are protected from wash-off by rain. Because of their persistence, it is important not to apply a systemic fungicide too close to harvest, as the fruit will become contaminated. Conversely Kumulus, which is a contact fungicide that remains on the surface of the plant tissue, is the only fungicide registered to control ELBS disease on saskatoons up to 1 d prior to the date of fruit harvest because it can be washed from the fruit after harvest has occurred (St-Pierre 1997; http://www.gov.mb.ca/agriculture/crops/fruit/bld01s01.html, last accessed on March 8, 2003).

Although Funginex and Kumulus were the first two fungicides registered to control ELBS disease on saskatoons, they do not control the disease as well as Topas 250E (Lange et al. 1998). Funginex, Topas 250E, and Kumulus each have a different active ingredient that adversely affects *E. mespili* growth and development on the saskatoon plant. Funginex utilizes the active ingredient triforine, to interfere with fungal growth. Lange et al. (1998) showed that Topas 250E suppresses both mycelial growth, by inhibiting cell wall formation, and conidiospore germination. Kumulus, which contains sulfur as its active ingredient, inhibits energy production within fungi.

Chemical management of disease should be scheduled before the exponential phase of saskatoon fruit growth to avoid applications too close to harvest (McGarry et al. 1998). Currently, it is recommended that Topas 250E be applied at the white tip, petal drop, and green fruit stages; the last application must be made not later than 38 days before harvest to avoid fruit contamination (Lange et al. 1998; St-Pierre 1997). Wright (pers. comm.), a Product Manager at Engage Agro Corporation, recommends that re-application of Topas 250E is necessary if rainfall occurs within one hour of application. Furthermore, it was suggested that current use of Topas 250E to control *E. mespili* on saskatoons is linked more to greater host tissue susceptibility while the plant is actively growing and developing than absolute longevity of the active ingredient propiconazole. This statement implies that the fungicide Topas 250E could be over-used, providing propiconazole is still active in the host plant when subsequent spray applications occur.

Overuse of fungicides can be damaging to the environment, consumers, and growers profits (Babcock et al. 1992). Chemical inputs present a dilemma for both farmers and society because these inputs have positive effects on the quantity and quality of farm products, while at the same time imposing costs on farmers, and posing potential health risks to humans and the environment. Engage Agro Corporation (Anonymous 2002) indicates that Topas 250E is registered to effectively control most diseases for a period of 21 d after each application. Therefore, knowing that anthesis in saskatoon plants lasts only 3.5 ±0.8 d (McKay 1973; St-Pierre and Steeves 1990) with the white tip and green fruit stages occurring shortly before and after this period, suggests that there is an over application of the fungicide based on currently recommended spray times (Lange et al. 1998). Furthermore, this fungicide application program assumes that ideal weather conditions for disease are present during this period to warrant a spray application. In a vineyard there are several advantages that are realized when the duration between fungicide applications is extended. For instance, fewer unnecessary chemicals are introduced into the vineyard environment, more access to the vineyard is gained, and the cost of chemicals and their application is decreased (Clarke 1996).

The timing and number of fungicide applications required for control will vary with geographic location depending on the time of primary inoculum release and host susceptibility during that time (van der Zwet 1990). For instance, release of inoculum is often environmentally driven (Madden et al. 1996), and subsequent infection is dependent upon the host's physiological condition (Baudoin 1986b). In saskatoons, it would be ideal to schedule fungicide applications according to environmental variables as they relate to inoculum release, and at times when the host is most susceptible to disease development. This would produce a more economical, environmental, and consumer friendly management protocol for ELBS disease on saskatoon.

3.1.6. Modeling Disease Development

There are many different types of forecasting models that can be used to study various biological systems. Krupa and Nosal (1989) used multivariate time series models to predict the damage caused by ambient sulfur dioxide on *Medicago sativa* L. A mechanistic model was created to explain sink priority regarding assimilate transport in phloem (Minchin et al. 1993; Minchin and Thorpe 1996). In a review paper, Whisler et al. (1986) listed and discussed more than 30 different crop growth simulation models. There are visual models such as YPLANT, which illustrate the architecture and development of woody plants helping to assess light capture and carbon gain of understory plants (Pearcy and Yang 1996). Such visual models may also be used to simulate the plant's response to disturbance. For example, Singh (2000) showed the regrowth of alfalfa plants after grazing.

When a biological system can be modeled with some degree of accuracy then the next logical step is to forecast or predict future development of that system so that management practices can be organized in advance of important events. Based on environmental parameters like temperature, there are phenological models that can be used to simulate insect development (Goldson et al. 1998), and plant development (Baker et al. 2001; Shaykewich 1995; Yan and Wallace 1998), as well as estimate crop yield (Raddatz et al. 1994). Shaykewich et al. (1998) and Zahner and Stage (1966) developed models to predict water deficits in potato crops and forest vegetation, respectively. Raddatz et al. (1996) developed a water use model based on phenological development of the potato plant. Such models can be used to schedule irrigation to potentially eliminate plant water stress and to help ensure maximum yields while conserving water (Shaykewich et al. 1998). It was shown that optimal irrigation scheduling during the exponential growth phase could help increase saskatoon fruit yield weight (McGarry et al. 1998).

Disease-forecast models, which are based on similar environmental parameters as required for plant growth, can be used to predict fungal inoculum dissemination (Pinkerton et al. 1998; Yang and TeBeest 1992), germination (van der Zwet and Stroo 1985), infection success (Gilles et al. 2001), and disease development (Papastamati et al. 2001). For example, Fernando et al. (1997) modeled head blight infection and dispersal gradients caused by *Gibberella zeae* (Schwein.) Petch from a single point of infection. van der Zwet and Stroo (1985) examined germination of *E. maculatum* conidia on *Photinia*, and Gilles et al. (2001) showed that a model could be constructed to describe the effects of temperature and leaf wetness duration on the development of light leaf spot on oilseed rape after infection by *P. brassicae* conidia. Similarly, Papastamati et al. (2001) developed a model to describe the progress over time of light leaf spot (*P. brassicae*) on leaves of winter oilseed rape. Models may even suggest a spray application to control the pathogen causing the disease. Lalancette and Hickey (1986b) developed a model for powdery mildew on apple based on plant growth, primary inoculum, and fungicide concentration.

The terms 'forecast' and 'prediction' can be used as synonyms according to the Webster's and Oxford dictionaries. Both deal with something future; they make a statement *ante factum* (Zadoks 1984). Confusingly, the forecast of future disease outbreak is primarily based on the observation of a past critical period like a weather event that causes dissemination of pathogen inoculum. With respect to pathogen infection and subsequent symptom development, such disease-forecast models are actually *post factum*. In other words, the disease-forecasting model typically identifies when disease symptoms will appear only after an infection event has already taken place. Predictive disease-forecasting models can be designed to predict disease development before symptoms are present, but they require knowledge of when inoculum is released and the time required for the pathogen to cause symptom development after initial infection.

The justification for constructing a disease-forecasting model is based on economic, environmental, and political factors (Royle 1985), and ultimately aims to reduce uncertainty about control decisions (Tait 1987). According to Royle and Shaw (1988), there are 3 main objectives for developing a disease-forecasting model. The first objective is to increase farm income by providing more efficient allocation of disease management resources. The second objective is to decrease the risk of large economic losses in crop value from disease, and the third is to decrease the amount of pesticide applied to crops, and thereby decrease the potentially harmful effects of the pesticide on the environment and human health.

Although some of the disease-forecasting models developed between 1935 and 1985 have provided useful information to growers and disease managers, they have been primarily developed without formal mathematical modeling (Fry and Fohner 1985). It has only been since the mid 1970's that mathematical models contributed to specific, practical forecast efforts. A disease-forecasting model should be a quantitative description of the relationship between host, pathogen, and environment, and should provide a foundation for calculating whether or not the application of a particular pesticide is justifiable (Zadoks 1984). Given that the amount of disease induced in a single season is dependent upon abundance and distribution of the pathogen, resistance of the host, and the time allowed for the host to interact with a pathogen in a particular set of weather conditions (Fry 1982b), an advanced disease-forecast modeling system should include a real-time weather monitoring system that combines a sound biological and physical understanding of how a pathogen interacts the host in their environment (Royle and Shaw 1988; Walker 1987).

The first stage of model development incorporates laboratory and field studies to predict the risk of disease and/or development of disease, based on monitoring key host, pathogen, and environmental variables. Hori and Kobayashi (1979) used a laboratory study to create a growth curve that illustrates how *E. mespili* colonies isolated from *Amelanchier*, can grow on media relative to increasing temperature. On a more complex level, models can be applied to disease progress curve data from the lab or field and express epidemic progress in terms of the absolute rate of change in disease intensity with respect to time (Nutter and Parker 1997). Pathogen population growth may follow one of five temporal growth regression equations (monomolecular, exponential, logistic, Gopertz or linear growth models) (Nutter and Parker 1997). Using the Nutter and Parker (1997) EPIMODEL software, raw and transformed data can be fitted to five of the temporal population growth models. The model with the highest coefficient of determination (R^2) value most accurately corresponds to actual disease progress, and thus is selected as the basis for the disease progress curve. Regression equations describing disease development can also be acquired from controlled studies assessing the effects of leaf wetness and temperature on pathogen germination, infection, and sporulation (Arauz and Sutton 1989; Gilles et al. 2001; Grove 2002; Mathieu and Kushalappa 1993; Monroe et al. 1997).

A disease-forecast model can be empirical or mechanistic in nature. Empirical models describe the relationships between variables without referring to any underlying biological or physical structure that may exist between the variables (Reynolds and Acock 1985). The most extreme form of an empirical model is the multiple-regression model, in which variables have been selected solely on the basis of their correlation to the variable being predicted (Hodges 1991b). As a greater understanding of the entire biological system is realized, a mechanistic, or rather a dynamic disease model for pathogen development can be created. Mechanistic models attempt to explicitly represent causality between all of the interacting environmental variables (Whisler et al. 1986). Several regression equations are used in a dynamic disease model, which is similar to a neural network model in that it tries to describe the interaction between the pathogen and each component of its environment (De Wolf and Francl 2000). The addition of detail is only limited by the effort needed to understand disease growth processes, by the capacity of computers to run the models, the amount of time needed to run the models, and by the amount of time needed to interpret the model results (Hodges 1991b). With the advanced state of computer technology it appears possible to add virtually unlimited amounts of detail to a disease model, thus increasing its dynamic nature.

Computers and real-time weather stations that are located in the crop canopy are essential tools for constructing dynamic disease-forecasting models. Several disease-forecast models, such as the tomato early blight model (Waggoner and Horsfall 1969), and potato late blight model (Bruhn and Fry 1981), have evolved from computer simulation models (Fry and Fohner 1985). One of the best known computer models in the United States is BLITECAST, a computerized forecast for potato late blight developed at Pennsylvania State University (Krause and Massie 1975). It was initially programmed on a large mainframe computer, but subsequently has become available on personal computers linked to weather data loggers (MacKenzie 1981). Other well-established forecast models have been programmed onto personal computers. For example, Castor et al. (1975) programmed the potential for Stewart's wilt of corn, and Jones et al. (1980) programmed an apple scab model for apple. Currently, advanced weather stations, like the ones produced by Adcon Telemetry (Adcon Telemetry, Klosterneuburg, Austria) measure environmental variables in real-time, and transmit this information to a client software program, which can then be used to help forecast disease development (Anonymous 1998). Adcon's software program, called addVantage graphs weather data and uses previously programmed models to predict disease epidemics for several crops including powdery mildew of grapes, scab of apple, and late blight of potato.

Most disease-forecast models developed to date incorporate just one, or in some cases two of the disease triangle components to predict disease development (Zadoks 1984). Disease-forecast models using disease symptoms as a basic component are rare, because in many cases it is too late to intervene when signs of the pathogen are already visible. When crop phenology is used as the only input in a forecast model, data are used to suggest the start of fungicide treatments when a specific plant development stage is reached. For example, a grower should start treating a potato crop for late blight when the green line stage is reached. As a single input, weather is the only basic ingredient used in disease-forecast models. However, Gwynne (1983) indicated that good agronomic and epidemiological intelligence should be integrated with weather data when developing a disease-forecasting model. The following sections describe models based on various types of input parameters.

Disease-forecast models can rely on estimates of inoculum production, and its efficacy. Inoculum production is an important component of disease-forecasting (Lalancette and Hickey 1986b). For instance, it would be of little value forecasting a possible outbreak of disease in the absence of inoculum, or more accurately, adequate inoculum. An assessment of inoculum production and efficacy can be useful for predicting disease severity for both monocyclic and polycyclic pathogens (Fry 1982b). The Stewart's wilt forecast model of corn is one such example where a lack of initial inoculum can lead to little or no disease development. Elliott and Poos (1934) explained the Stewart's wilt forecast model by showing that the bacterium, which causes the disease, overwinters in the corn flea beetle. Where efficacy refers to the survival of the corn flea beetle, the forecast model estimates the relative size of the beetle population, which in a cold winter does not survive well, and as a result disease levels decrease in the next growing season. For *V. inaequalis*, initial inoculum infection success is very important for scab development on apple trees because there are only a few secondary cycles of pathogenesis. The pathogen relies on initial inoculum in the spring to infect young leaves and fruit, which are highly susceptible to infection at this time (Boone 1971). If ideal weather conditions are not present, inoculum viability and infection success is subsequently diminished. When developing a disease-forecast model that relies on an assessment of initial inoculum, the grower must be willing to assess disease severity in the crop (Royle and Shaw 1988).

The accuracy of a forecasting model can be further improved if it includes information regarding the time when inoculum is released in relation to environmental factors. One specific example is the disease-warning model for apple scab. This model is based on the measurement of precipitation, which promotes inoculum release (MacHardy and Gadoury 1986), and leaf wetness durations and temperature after the precipitation event, which is required for infection (MacHardy and Gadoury 1989). Many different factors that affect the release of inoculum from different pathogens have been identified, including rainfall and free water (Brook 1969a; Ingold 1971; Luley and McNabb 1989; Rowe and Beute 1975; Sutton 1981), temperature (Bertrand and English 1976; MacHardy and Gadoury 1986; Tate 1979), humidity (Gregory and Stedman 1958; Linderman 1974; Sutton 1981), light (Brook 1969b; Hayes 1980; Leach 1980; Luley and McNabb 1989;

McCoy and Dimock 1973), and vibration (Leach 1980). Relatively little is known about the timing of *E. mespili* inoculum release on saskatoon relative to environmental conditions.

When a host plant is exposed to a particular pathogen, it can be described as having some level of resistance or susceptibility. Hosts that have high levels of disease resistance may show few to no visible symptoms, while highly susceptible hosts may completely succumb to the pathogen (Dickinson and Lucas 1982). Nelson (1973) defined disease resistance as an active, dynamic host response to a parasite. Both Davidson (1989), and Lange and Bains (1994) suggested that none of the commercially grown saskatoon cultivars showed resistance to *E. mespili* infection. However, Ronald et al. (2001) qualitatively and quantitatively rated *E. mespili* infection among 17 saskatoon cultivars and revealed differences in disease resistance. The saskatoon cultivars 'Parkhill', 'Regent', and 'Success' showed a greater degree of resistance to *E. mespili* than cultivars 'Honeywood', 'Smoky', 'Pearson II', and 'Forestburg'. Unfortunately, the 'Parkhill', 'Regent', and 'Success' cultivars are not currently used in commercial saskatoon orchards, because the quality and quantity of their fruit is not as good as the more commonly used cultivars 'Honeywood', 'Northline', 'Smoky', and 'Thiessen'. This suggests the need for a breeding program that would integrate the disease resistance expressed in 'Parkhill', 'Regent', and 'Success' with the superior fruit quality and quantity of the more commonly used orchard cultivars.

Young plant tissue is often more susceptible to disease than older fully expanded leaves (Baudoin 1986b; Jacobs et al. 1996; Ronald et al. 2001; Seem and Szkolnik 1978). Young expanding leaves of *Photinia* X *fraseri* Dress. are highly susceptible to *E. mespili*, while mature, fully expanded leaves are much less susceptible to infection (Baudoin 1986b). Jacobs et al. (1996) showed that ELBS disease severity could be 10 to 30 times greater on young *Photinia* leaves. Ronald et al. (2001) also found that young saskatoon leaves are more susceptible to *E. mespili* infection than older leaves. Young saskatoon leaves are also more susceptible than older leaves to other pathogens like *Gymnosporangium nelsonii* Arth., which causes rust disease (Kabaluk and St-Pierre 1992). A model that incorporates the interaction between plant growth parameters and disease susceptibility has the potential to predict disease development more accurately (Lalancette and Hickey 1986a).

Resistance based on increasing leaf age implies that the chemical and (or) physical nature of older leaves reduces symptom development. Wetzstein and Sparks (1983) noted that higher levels of resistance in older leaves of *Carya illinoensis* Wang. were associated with low trichome density and longevity, high phenolic content in the mesophyll, and thick cuticles. As a leaf begins to age, waxes may build up on its surface. Waxes on leaf and fruit surfaces form a water repellent layer and thereby prevent the formation of a film of water in which inoculum might be present (Agrios 1997). Knowing that *E. mespili* primarily infects plants like *Photinia* through direct cuticle penetration on the abaxial side of the leaf, a thicker cuticle would provide a superior physical barrier to infection. However, it should be noted that *E. mespili* can still circumvent the thick cuticle and cause infection in older leaves by entering through guard cells or stomata

(Baudoin 1986b). Ronald and St-Pierre (2002) found no evidence where *E. mespili* conidia germinate and directly penetrate saskatoon leaves through natural openings or wounds, but indicated these methods of entry cannot be ruled out.

The number of secondary cycles a pathogen can produce in a growing season, and the environmental conditions that affect each cycle are important factors to consider in disease-forecasting dynamics. For example a forecast model for the polycyclic pathogen *Uncinula necator* (Schwein.), which causes powdery mildew on grapes, should incorporate the rapidity of secondary cycles or the amount of secondary inoculum being produced (Fry 1982b). Similarly, *E. mespili* can also undergo many reproductive cycles during a growing season, providing weather conditions are ideal for short but specific periods during each reproductive stage. Sinclair et al. (1987) and van der Zwet and Stroo (1985) showed that the gestation time of *E. maculatum* is relatively short (2 to 4 wk), and therefore management input is required at a low level of visible disease for adequate suppression (Fry 1982b). This is because little time is required to rapidly increase the number of infection points, leading to the production of even more inoculum, and ultimately elevating disease levels.

Disease-forecasting models have been designed to incorporate epidemiological components of the pathogen with past and present environmental conditions. Fry and Fohner (1985) show several models that use an estimate of initial inoculum, weather factors, and in some cases both inoculum and weather to predict disease development on various crops. The powdery mildew model for grapes, which was developed by Thomas et al. (1994), predicts disease development based on ascospore and conidium availability in the spring when the vineyard temperature exceeds 10°C and leaves are moist for more than 12 h during the day (Clarke 1996).

Once a disease-forecasting model has been constructed, its predictive ability must be assessed before growers can implement on a large scale. Disease-forecast models are validated by comparing observed disease symptom development from laboratory and or field evaluation trials against predicted values that have been generated by a regression model (Asher and Williams 1991). The predictive ability of a model can be measured by visually comparing figures illustrating disease symptom development relative to predicted values of disease increase, or by analysis of correlation coefficients between these two variables. Gilles et al. (2001) illustrated the predictive ability of a *P. brassicae* model by comparing the observed percent leaf area that was undergoing sporulation against predicted values from the model. Their results showed that the model accurately predicted observed disease development in the field for periods up to 37 and 45 d from the prediction date, but became less accurate after this time. De Wolfe and Francl (2000) evaluated disease-forecasting models for tan spot and stagonospora blotch on hard red spring wheat in the field using artificial neural networks and found that the models correctly classified 82 to 84% of observed disease severity, respectively. Grove (2002) tested leaf wetness duration and temperature models to predict the number of *Wilsonomyces carpophilus* (Lev.) lesions per unit area of leaf surface on cherry and peach in a controlled environment study, and produced R^2 values of 0.80 and 0.84, respectively. In sugar beet crops, Asher and Williams (1991) found that temperature and precipitation accounted for 95% of the variation in powdery mildew development. Pfender (2003) created a model based on wetness degree-hours that explained 80% of the variability in the infection success of *P. graminis* subsp. *graminicola* on perennial ryegrass seed crops. Models that have a high R^2 value are considered to have reliable predictive ability, as they help to explain most of the variation expressed between the observed and predicted variables. After analysis of laboratory and field studies, models must be validated across a variety of microclimates over a number of years (http://www.ipm.ucdavis.edu/DISEASE/california_pestcast.html, last accessed on March 8, 2003). For example, Bruhn and Fry (1981) used three growing seasons to validate a model for potato late blight on four potato cultivars, and Pfender (2003) used three independent sets of data to test a model for stem rust on perennial ryegrass seed crops.

3.2 Forecasting Phenological Development Of *Amelanchier alnifolia*

Phenology is defined as a branch of science dealing with the relations between climate and periodic biological events. Dethier (1978) suggested that there is a direct relationship between the phenological development of a plant and past and present weather conditions. For example, temperature is particularly important for governing plant development (Eggert 1960; Richardson et al. 1975; van den Brink 1974), and it was found to be the only environmental variable that has any significant effect on the phenological development of peach fruit (Smith 1985). The amount of heat required for a plant species to reach each stage of phenological development can be used as a measure of physiological time (Strickler 1998). Physiological time provides a universal reference for the development of an organism based on heat unit accumulation. Anstey (1966) suggested that the best way to monitor the phenological development of a plant that exhibits preformed growth, like the saskatoon, is to measure the total amount of heat units which are required to reach the phenological stages of flowering and fruit harvest, and the cessation of axillary vegetative shoot expansion in the growth season after organ initiation.

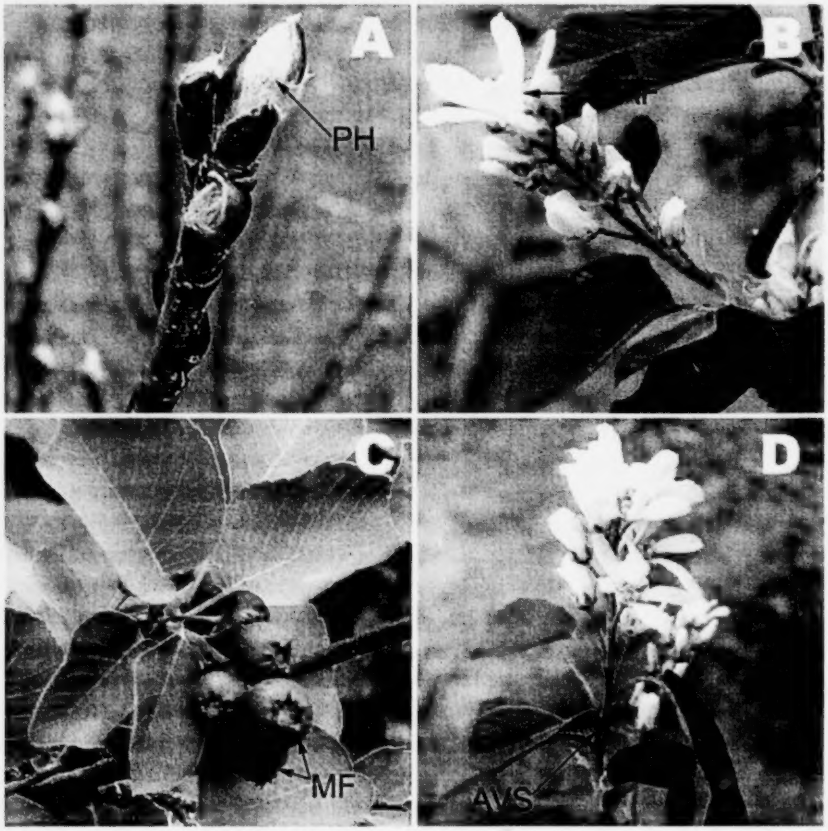
A precise and reliable phenological forecasting model that will support the orchard management practices of pesticide application and fruit harvest is currently a limiting factor affecting saskatoon fruit production. In other fruit crops such as apple, pear, cherry, peach, and apricot, phenological modeling based on growing degree-day (GDD) heat unit accumulation has been used to predict the date of full bloom and has provided growers with an advanced warning system for spray and thinning programs (Anstey 1966; Sisler and Overholser 1943). For each 24 h period, GDD heat units are calculated by subtracting the minimum temperature at which the plant can still grow and develop from the mean daily temperature (Wolf et al. 1986). Knowing in advance when saskatoon plants will flower will promote the precise timing of pesticide applications based on current spray recommendations (Lange et al. 1998; St-Pierre 1997). Moreover, as the physiological age of saskatoon leaves increase, their susceptibility to fungal pathogens like *Entomosporium mespili* (D.C.) Sacc. decreases (see Chapter 4; Ronald et al. 2001). Therefore, knowledge of the physiological development of vegetative tissue could be important for determining the need for fungicide applications as the plant develops. The GDD model has also been used to predict the date of fruit harvest after the date of flowering for several varieties of apples, pears, peaches, and cherries (Anstey 1966; Smith 1985; Tukey 1942). Friesen (1986) used the same GDD model, calculated with a base temperature of 5°C, to predict the date of fruit maturity for various periods after flowering in saskatoons. With an accurate prediction model for fruit harvest date, growers will be able to schedule harvest operations in advance to ensure that the highest quality and quantity of fruit is available at the time of harvest. Inability to conduct harvest operations in advance of peak ripeness can lead to a harvest of overripe fruit which does not store well and can make processing difficult (St-Pierre 1997). An accurate phenological forecasting model that predicts flowering, fruit harvest, and the cessation of axillary vegetative shoot growth would strengthen the saskatoon industry by helping growers plan orchard management activities.

There are various ways of expressing the amount of heat that is useful for plant development. Although the GDD system is the simplest and historically probably the first (Eddy 1977), the accuracy of the GDD model is impaired by shortcomings in its design (Shaykewich 1995). In an attempt to improve upon the accuracy of the GDD model, Sands et al. (1979) created the physiological day model (P-Day) to predict various stages of potato plant development based on a minimum, optimum, and maximum temperature required for plant growth and development. The P-Day model has proven to be useful in overcoming the inaccurate linear response to temperature and overestimation of heat units in the GDD model (Shaykewich 1995). Therefore, the main objectives of this part of the study were: 1) to modify the P-Day model to suit saskatoon; and 2) to compare the precision of a saskatoon P-Day model against the GDD model and a Julian day system for predicting the dates of flowering, fruit harvest, and the cessation of axillary vegetative shoot expansion from the date of bud break.

3.2.1. Materials and Methods

The phenological development of mature 'Smoky' saskatoon orchards, located in Winnipeg and Carman, Manitoba, were monitored during the growing seasons of 1999, 2000, and 2001. In each year, field monitoring began on Julian day 105 while the plants were still dormant and continued until fruit harvest was complete. The important phenological stages had to be properly defined so that their relationship with physiological time could be determined. Phenological development of the saskatoon floral bud has been previously defined in various studies (Friesen 1986; St-Pierre 1997; Sumner et al. 1999). Because it is comprehensive and widely used by growers, the phenological scale of St-Pierre (1997) was used to record the Julian dates of bud break and flowering in the field. A bud was considered dormant until it began to swell, separating its outer scales and revealing pubescent hairs that had a silvery appearance (Fig. 3.1A). An orchard was considered to have reached the stage of bud break when more than 50% of 10 floral buds from each of 10 randomly sampled plants began to swell and reveal their silver pubescent hairs. Once the petals of the apical flower were completely unfolded to expose the reproductive organs, the inflorescence was considered to have reached the flowering stage of phenological development (Fig. 3.1B). The date of flowering was identified as the time when more than 50% of the inflorescences arising from the randomly sampled reproductive buds had reached the phenological stage of flowering. The 9-stage maturity class index based on saskatoon fruit color was used to determine the first date of fruit harvest (Rogiers and Knowles 1997). Although maximum fruit weight is not realized until the ninth stage of fruit ripening, it was decided that harvesting activities should commence when the fruit reach the eighth stage of development. This would improve storability and processing functionality of the fruit. Therefore, the first date of fruit harvest was identified when more than 50% of the apical fruit on the sampled inflorescences reached stage 8. Fruit that have reached stage 8 are described as being very dark red with deep purple hues (Fig. 3.1C). The date of fruit maturity was not correlated with a measured index of fruit quality aside from fruit color.

Growth of vegetative tissue on saskatoon plants was determined by measuring the length of the axillary vegetative shoot (St-Pierre and Steeves 1990), which is located at the base of a reproductive saskatoon short shoot (Fig. 3.1D). The cessation of vegetative tissue growth was determined by fitting regression curves to the expansion of axillary vegetative shoots. A point on the x-axis, which was perpendicular to the apex of the regression curve, was used to identify the date that the axillary shoots ceased expansion. All of the leaves arising from the axillary vegetative shoots were near maturity when shoot expansion was complete.



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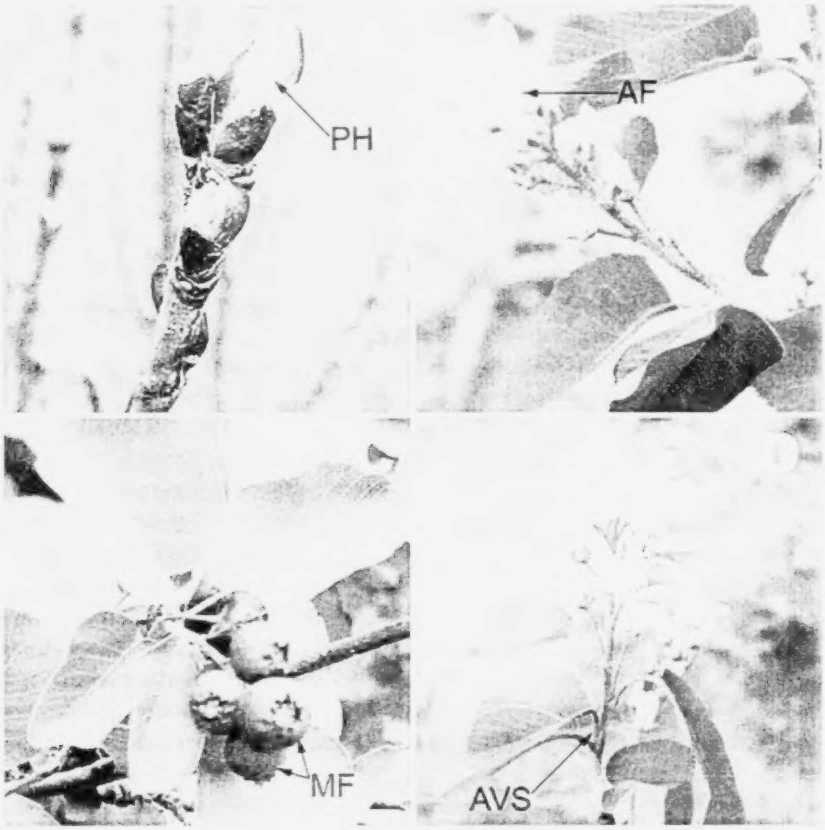

Figure 3.1 Key phenological stages of saskatoon floral and fruit development. **A.** A terminal floral bud on a saskatoon plant that has reached the phenological stage of bud break. The swelling of the bud and separation of the outer scales to reveal silver colored pubescent hairs (PH) characterizes bud break. **B.** An inflorescence that has reached the phenological stage of flowering. The inflorescence was considered to be at the flowering stage when the petals of the apical flower (AF) are completely unfolded to expose the reproductive structures of the flower. **C.** Saskatoon fruit that are mature and ready for harvest. Mature fruit (MF) that is ready for harvest is very dark red with deep purple hues. **D.** A flowering shoot with a terminal inflorescence and an axillary vegetative shoot (AVS) arising from the apex of the lowermost leaf.

To create a phenological model for saskatoons (hereafter known as SASK-Day) it was necessary to modify the minimum, optimum, and maximum temperature parameters of the potato P-Day model to better suit the critical temperatures that affect saskatoon plant growth and development. These data were obtained from another study that examined saskatoon stem diameter growth over a 14 d period at various temperatures. These data suggest that 20°C is optimum and 30°C is the maximum temperature affecting saskatoon plant growth. Twenty degrees Celsius was selected as the optimum temperature because that temperature treatment had a lower variation in growth compared to the 15°C treatment, and it was closer to the 21°C optimum temperature used in the original P-Day model (Sands et al. 1979). Junttila et al. (1983) previously identified the minimum temperature required for saskatoon plant growth as 5°C.

The SASK-Day model assumes that the rate of plant development is zero at temperatures less than the minimum temperature; plant development increases linearly between the minimum and optimum temperatures, then decreases linearly to zero at the maximum temperature. The modified P-Day equation, which was used to calculate daily accumulations of SASK-Day heat units, is illustrated in equation [7]:

[7] $$\text{SASK-Day} = (1/24)\ \mathrm{X}\ [5\ \mathrm{X}\ P(T_1) + 8\ \mathrm{X}\ P(T_2) + 8\ \mathrm{X}\ P(T_3) + 3\ \mathrm{X}\ P(T_4)]$$

where SASK-Day represents the daily accumulation of heat units, T_1 is equal to a 24 h minimum temperature (T_{min}); T_2 is derived from equation [8]:

[8] $$T_2 = ((2\ \mathrm{X}\ T_{min}) + T_{max}) / 3$$

T_3 is derived from equation [9]:

[9] $$T_3 = (T_{min} + (2\ \mathrm{X}\ T_{max})) / 3$$

and T_4 is equal to the daily maximum temperature (T_{max}). The denotation for P is a heat unit value that is calculated differently for different values of temperature (T): 1) equal to zero when T is less than the minimum temperature required for plant development (5°C); 2) calculated according to equation [10] when T is greater or equal to 5°C and less than the optimum temperature required for plant development (21°C):

[10] $$P = k\ \mathrm{X}\ (1 - [(T - 20)^2 / (20 - 5)^2]$$

3) calculated according to equation [11] when T is greater than or equal to 20°C and less than the maximum temperature required for plant development (30°C):

[11] $$P = k\ \mathrm{X}\ (1 - [(T - 20)^2 / (30 - 20)^2]$$

and 4) is equal to zero when T is greater than or equal to 30°C. The denotation for k is a constant of 10.

The accumulation of GDD heat units required for saskatoon plant growth and development were calculated. The daily accumulation of GDD heat units was determined by subtracting a base temperature from the daily mean temperature. The mean temperature was calculated by averaging the daily maximum and minimum temperatures. As in the SASK-Day model, 5°C was used as the base temperature required for saskatoon plant growth and development. The following equation (Friesen 1986) was used to calculate GDD heat units for saskatoon:

$$[12] \qquad GDD = [(T_{max} + T_{min})/2] - T_{base}$$

where GDD represents the daily accumulation of heat units, T_{max} is the daily maximum temperature; T_{min} is the daily minimum temperature; and T_{base} which is the base temperature. The daily GDD heat units were summed to provide an estimate of plant development.

One Adcon Telemetry (Adcon Telemetry, Klosterneuburg, Austria) weather station was positioned in a central location in both the Winnipeg and Carman orchards. Each weather station measured temperature 15 cm in from the outer edge of the plant canopy on a randomly selected plant every 15 min between the months of April and August. The temperature data were transmitted from the field via a relay network comprised of FM radio signals and telephone connections to a server computer, which processed the data and made it available for analysis. Temperature readings were used to calculate daily heat unit accumulation using the SASK-Day and GDD models every 24 h cycle, which started at 12:00 am on the date of bud break in each orchard. Using each model, the sum of daily heat units required to reach the phenological stages of flowering, fruit harvest, and the cessation of axillary shoot expansion from the date of bud break between years 1999 to 2001, were recorded. The mean number of heat units and the standard deviation period for each phenological event were calculated from combined site and year data.

In order to compare the accuracy of the SASK-Day model and GDD model against each other and with the Julian day system for predicting phenological development, the heat unit standard deviation period for each of the models was converted into calendar days. For each heat unit model an average daily heat unit value was first calculated, which was representative of the time period around each phenological event, by averaging the daily heat unit values from 2 d before and after the dates of flowering, fruit harvest, and completed axillary vegetative shoot expansion at each site and year. The averaged daily heat units for each model, which were calculated for each phenological event, were then divided into their respective standard deviation periods to obtain a standard deviation period that was represented in days. Once the standard deviation period for each heat unit model was converted into a period of days, it was possible to identify the most accurate model by selecting the one with the lowest standard deviation value.

3.2.2. Results

During the course of this study, the date of bud break ranged between Julian day 109 and 120, the date of flowering ranged between Julian day 126 and 137, and the date of fruit harvest ranged between Julian day 186 and 194. Different axillary vegetative shoot growth curves for the sites and years are illustrated in Fig. 3.2. The axillary vegetative shoots grew considerably longer in the Winnipeg orchard during the 1999 and 2000 field seasons. Nevertheless, the time of shoot growth cessation was similar at all sites and years. The high R^2 values for the polynomial regression lines showed that they were a good representation of growth over time, and therefore it was possible to confidently identify the asymptote of each growth curve. As determined from the growth curves, axillary vegetative shoots ceased to expand between Julian day 173 and 183.

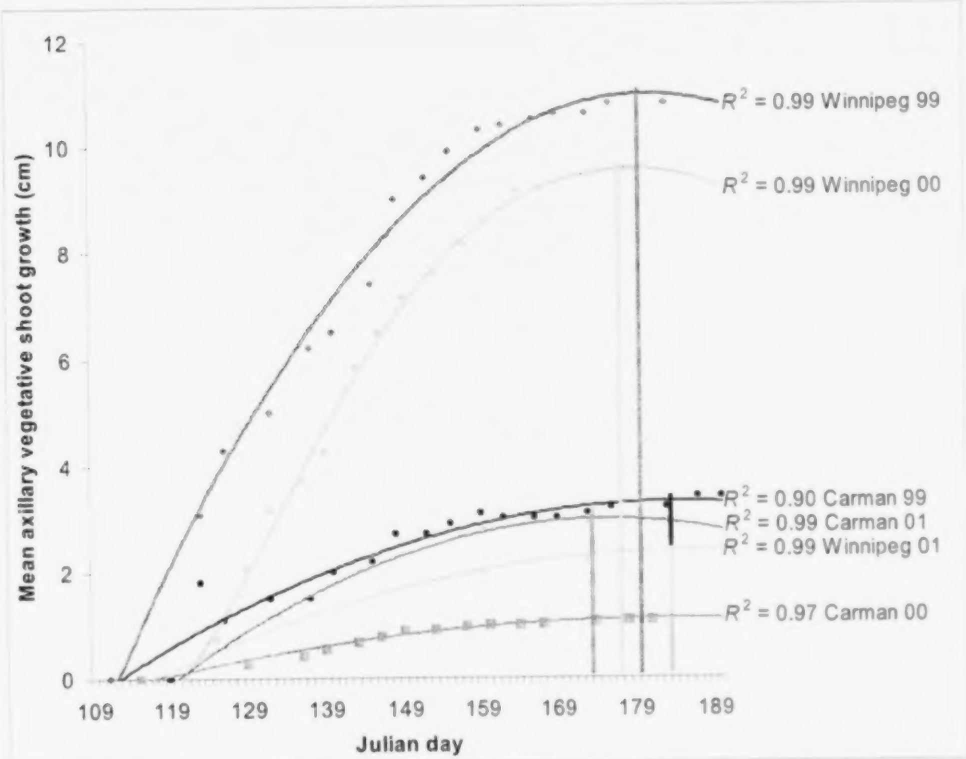

Figure 3.2 Mean axillary vegetative shoot growth at the Winnipeg and Carman saskatoon orchards for the years 1999 to 2001. Coefficients of determination (R^2) indicate the goodness of fit of each polynomial regression curve. Lines perpendicular to the x-axis indicate the asymptote of each growth curve and were used to identify the date at which the axillary vegetative shoot ceased to expand.

The SASK-Day and GDD heat units required to reach the stages of flowering, fruit harvest, and completion of axillary vegetative shoot expansion from the date of bud break are illustrated in Tables 3.1 and 3.2, respectively. The mean number of heat units (± standard deviation) required to reach flowering, fruit harvest, and the completion of axillary vegetative shoot expansion from the date of bud break were 112.1 (±13.5), 515.9 (±21.1), and 441.4 (±42.0), respectively, using the SASK-Day model, and 151.7 (±20.0), 665.1 (±73.0), and 628.1 (±72.2), respectively, using the GDD model. The mean number of Julian days required for reaching flowering, fruit harvest, and the cessation of axillary vegetative shoot expansion from the date of bud break were 20 (±3.0), 76 (±5.0), and 67 (±5.9), respectively (Table 3.3).

When the standard deviation values were converted to days (Table 3.4), the largest difference was evident for fruit harvest. The SASK-Day model had the lowest standardized standard deviation values for each phenological event, followed by the GDD model and then Julian days. Except for flowering there was minimal difference between GDD and Julian days.

Table 3.1 Number of SASK-Day heat units required to reach the phenological stages of flowering, fruit harvest, and the cessation of axillary vegetative shoot expansion from the date of bud break at the Winnipeg and Carman saskatoon orchards during 1999, 2000, and 2001, along with the mean and standard deviation values for combined sites and years. The SASK-Day values are rounded to the nearest first decimal place.

		Phenological event		
Year	Location	Flowering	Fruit harvest	Shoot growth cessation
1999	Winnipeg	96.5	527.1	465.8
2000	Winnipeg	137.0	550.3	443.1
2001	Winnipeg	109.4	509.4	492.4
1999	Carman	107.9	490.7	440.7
2000	Carman	113.4	517.1	440.0
2001	Carman	108.2	500.5	366.5
Mean		112.1	515.9	441.4
Standard Deviation		13.5	21.1	42.0

Table 3.2 Number of GDD heat units required to reach the phenological stages of flowering, fruit harvest, and the cessation of axillary vegetative shoot expansion from the date of bud break at the Winnipeg and Carman saskatoon orchards during 1999, 2000, and 2001, along with the mean and standard deviation values for combined sites and years. The GDD values are rounded to the nearest first decimal place.

		Phenological event		
Year	Location	Flowering	Fruit harvest	Shoot growth cessation
1999	Winnipeg	158.8	626.9	667.3
2000	Winnipeg	183.1	599.2	568.7
2001	Winnipeg	142.5	726.7	702.5
1999	Carman	133.0	618.2	674.3
2000	Carman	161.4	636.3	642.2
2001	Carman	131.1	783.6	513.7
Mean		151.7	665.1	628.1
Standard Deviation		20.0	73.0	72.2

Table 3.3 Number of Julian days required to reach the phenological stages of flowering, fruit harvest, and the cessation of axillary vegetative shoot expansion from the date of bud break at the Winnipeg and Carman saskatoon orchards between the years of 1999 and 2001, along with the mean and standard deviation values for combined sites and years.

		Phenological event		
Year	Location	Flowering	Fruit harvest	Shoot growth cessation
1999	Winnipeg	16	77	70
2000	Winnipeg	24	83	68
2001	Winnipeg	18	70	70
1999	Carman	19	72	70
2000	Carman	22	80	66
2001	Carman	18	73	55
Mean		20	76	67
Standard Deviation		3.0	5.0	5.9

Table 3.4 Comparison of SASK-Day, GDD, and Julian day standard deviation values, which are expressed in days for the predicted dates of flowering, fruit harvest, and the cessation of axillary vegetative shoot expansion. Values are rounded to the nearest first decimal place.

	Phenological event		
Model	Flowering	Fruit harvest	Shoot growth cessation
SASK-Day	±2.1	±2.8	±5.1
GDD	±2.5	±4.8	±5.8
Julian day	±3.0	±5.0	±5.9

3.2.3 Discussion

To create the SASK-Day model in this study, 20°C was assumed to be the optimal temperature and 30°C the maximum temperature required for saskatoon plant growth and development. However, the selection of these temperatures was based on a limited number of plants, measured over a limited time period. Therefore, more research is required to confirm these critical temperatures. Despite this limitation, the SASK-Day model produced the most accurate prediction of flowering, fruit harvest, and the cessation of axillary vegetative shoot expansion from the date of bud break, producing the lowest standardized standard deviation values. The next best method for predicting saskatoon plant growth and development from the date of bud break was the GDD model followed closely by the Julian day system. In another study, Friesen (1986) showed that the GDD model produced an R^2 value of 0.96 when predicting the date of fruit harvest beginning 40 d after flowering using a quadratic regression equation. This model accurately predicted harvest within ±1 d for the four years in which it was developed. Although the predictive ability of the models in the present study were not specifically tested using regression analysis, the SASK-Day model more reliably identified the date of fruit harvest earlier in the season than Friesen's (1986) model. Moreover, compared to other models based on standard deviation for predicting flowering in apple, pear, cherry, peach and apricot (Anstey 1966), the SASK-Day model had standard deviation values 2 to 5.4 d lower.

Results of this study are logical considering the GDD model has been shown to be more precise than the Julian day system for predicting the harvest date of processing tomatoes (Wolf et al. 1986), and that the GDD model is limited by a number of practical problems (Shaykewich 1995), which the P-Day model helps to overcome. For instance, the GDD equation assumes that the growth response of the plant is linear over an entire temperature range, and as a result it can overestimate the number of heat units accumulated if the temperature is above the optimum temperature required for plant growth and development. Essentially, the GDD system assumes there is a linear relationship between plant growth and temperature with no maximum. This does not correspond to what actually occurs in nature (Morris et al. 1980), and therefore leads to the conclusion that the SASK-Day model, which incorporates a specific minimum, optimum, and maximum temperature for saskatoon plant growth, is more accurate for predicting phenological development.

In conclusion, a SASK-Day heat unit model was created that more accurately predicts the dates of flowering, fruit harvest, and the cessation of axillary vegetative shoot expansion than the GDD and Julian day systems. The SASK-Day model will be used to forecast the dates of flowering and fruit harvest as early as possible in the growing season, and model axillary vegetative shoot growth. Because the phenological stage of flowering can be used as a reference point for the commencement of pesticide application, identifying the date of flowering in advance will allow for the advanced planning of Decis 5.0EC applications to control insect pests like apple curculio, hawthorn weevil, lygus bug, saskatoon bud moth, saskatoon sawfly, and tarnished plant bug, and Topas 250E applications to control ELBS disease on saskatoon (St-Pierre 1997). Growers will be able to optimize harvest efforts by planning harvest operations early in the growing season with a phenological model that predicts the date of fruit harvest. The heat units required for the cessation of axillary vegetative shoot growth will be used to create a negative linear equation in Chapter 6 that models decreasing susceptibility of the saskatoon plant to *E. mespili* relative to increasing physiological time. This regression analysis will provide a measurement of plant susceptibility at various points throughout the growing season. The measurement for plant susceptibility will be utilized as part of a dynamic disease-forecasting model that will be used to calculate disease pressure in an orchard and subsequently suggest timely fungicide applications to help control ELBS disease. Due to the systemic nature of the Topas 250E fungicide, the SASK-Day model for fruit harvest date will also be incorporated into the dynamic disease-forecasting model to prevent fungicide applications from occurring less than 38 d before the predicted harvest date, and thus prevent fruit contamination. The SASK-Day model must still be validated for several years at different locations before it is considered accurate and incorporated into the production system of saskatoon growers. Later in Chapter 6 the accuracy of the SASK-Day phenological model for predicting flowering and fruit harvest will be assessed when it is integrated with historical weather data.

3.3. Leaf Wetness Duration And Temperature Required For The Development Of *Entomosporium mespili* On Saskatoon Leaves Under Controlled Conditions

Specific weather conditions are often important in causing disease levels to increase within a crop (Jones 1986). Empirically determined relationships between environmental factors and disease have been used to time fungicide sprays for many diseases (Grove 2002; Thompson and Jenkins 1985). Therefore, management of ELBS disease should depend on knowledge of the environmental conditions that promote pathogen infection and development. In the case of saskatoon, field observations suggest that *E. mespili* development is promoted by warm, moist conditions (Davidson 1990; Horie and Kobayashi 1979; Sinclair et al. 1987; St-Pierre 1997). Lange and Bains (1995) indicated that heavy rainfall is the most important environmental factor contributing to *E. mespili* increase in saskatoon orchards. Baudoin (1986a) showed that the optimum temperature for *E. mespili* infection on *Photinia* X *fraseri* Dress. was 20°C, and that disease levels increased as the duration of leaf wetness increased. However, the specific leaf wetness and temperature conditions required to promote *E. mespili* development on saskatoon plants are not known. Moreover, a recent study has indicated that disease development depends on leaf age, in that young saskatoon leaves are more susceptible to infection than older leaves (Ronald et al. 2001), and therefore, the effect of leaf age on disease development in relation leaf wetness duration and temperature is also not understood. In this context, the objectives of this study were: (1) to quantify the leaf wetness duration and temperature required for causing ELBS disease on saskatoon plants, and develop a regression equation for this relationship; and (2) to identify the leaf wetness and temperature conditions that provide optimal lesion development on young versus old leaves.

3.3.1. Materials and Methods

Dormant, two-year-old saskatoon plants of the cultivar 'Smoky', derived from tissue culture, were obtained from D'nA Gardens in Red Deer, Alberta. Each plant consisted of one main stem that ranged in length from 10 to 25 cm. The plants were kept dormant in cold storage (0°C, 30% RH) for 75 to 105 d after arrival. There were fewer growth rooms available than temperature treatments, so the experiment was conducted over time, resulting in one set of inoculations per leaf wetness duration and temperature treatment. Groups of plants selected for each temperature treatment were taken out of cold storage in 14 d intervals to ensure that each group was at the same stage of phenological maturity for each temperature treatment. Each plant was placed in a pot filled with a mixture of soil, sand, and peat moss (1:1:1, v:v). Granular fertilizer (40 g of 11-53-0) was added to the mix at the time of planting. Subsequent applications of 20-20-20 (N-P-K) were applied during watering at 2 wk intervals after planting. The plants were grown in a greenhouse maintained at 20 ±2°C during the light period (16 h) and at 18 ±2°C in the dark (8 h). After planting, the branches of each plant were sprayed once a week with a 250-ppm gibberellic acid solution to stimulate bud break (Sumner et al. 1999). This planting procedure helped to ensure the plants were leafed-out and ready for inoculation approximately one month after the planting date.

Leaves and fruit that were heavily infected with *E. mespili* were collected from a mature saskatoon orchard located near Winnipeg, Manitoba in September of 1998 and 1999, and used as a source of inoculum for the first and second repetitions of the study, respectively. This material was air dried and stored in a sealed plastic container at 5°C until needed. Infected leaves and fruit were allowed to acclimate at 20°C for three days before they were used to prepare the conidial suspension. About 40 leaves and five fruits were placed in a beaker containing 200 mL of distilled water. The suspension was vigorously agitated with a stir rod for 20 min to stimulate release of the conidia from the acervuli. Plant debris was filtered from the suspension and the number of conidia per milliliter was determined using a hemacytometer. The suspension was then diluted to approximately 10^5 conidia/mL. Before the plants were inoculated, two drops of Tween 20 were added to the suspension to ensure good leaf surface contact. The conidial suspension was agitated continuously, until it was applied to the plant, to ensure that the conidia were evenly distributed in the suspension.

Plants were inoculated with an air atomizer operated at 20 psi (1 psi = 6.895 kPa) until runoff. Inoculated plants were randomly placed in a mist chamber assembled from polyethylene plastic sheets and positioned within a growth room set at 18°C similar to the temperature used by Baudoin (1986a). Continuous mist was provided from a mist humidifier located to one side of the humidity chamber and above the plant canopy. In the first repetition of the study, two leaf wetness periods were tested (12 and 24 h). Given the limitation in growth room resources, 24 h was selected as the maximum period of leaf wetness in accordance with other crop disease studies (Grove 2002; Monroe et al. 1997). Moreover, leaf wetness duration in Manitoba, which is one of the wetter areas of the Prairies, typically remains below 24 h, based on field monitoring data (data not shown). After analyzing the results, two leaf wetness periods (0 and 6 h) were added to the second repetition of the study. The addition of two leaf wetness periods in the second repetition of the study was done to better assess the hours of leaf wetness required for infection. After removal from the mist chamber, each pot was placed in an acetate sleeve to prevent interplant contact, and in a dish to trap excess water runoff after watering. The plants were then moved to another growth room preset to the desired temperature treatment and constant relative humidity of 30%.

Temperature treatments of 15, 20, 25, and 30°C were used in the first repetition of the study, and a fifth temperature treatment of 10°C was included in the second. The 10°C temperature treatment was added in the second repetition in an attempt to identify the minimum temperature at which the pathogen could still cause infection. The growth room provided a constant temperature and 16 h day and 8 h night over the 14 days of temperature-treatment period. Light for the 16 h day period was supplied from both fluorescent and incandescent bulbs. Each plant was kept adequately watered during the entire study by applying water directly to the soil surface. Plants were moved randomly within the growth room every second day to minimize the impact of variable conditions within the growth room itself. Each temperature treatment had control plants that did not receive inoculation or a period of leaf wetness. Temperature treatments were randomized within each study.

Plants were examined for leaf lesions every second day after the first day of each 14-day temperature-treatment period. After the 14-day temperature-treatment period, 5 leaves from the top and 5 from the bottom half of each plant were randomly sampled to compare susceptibility of the young leaves found near the apex of the plant, with physiologically older leaves found near the base of the plant. Disease severity at the end of each trial was determined by calculating the percent-infected leaves per plant (PINFL):

[13] $$\text{PINFL} = (X/Y)*100$$

where X is the number of infected leaves on a plant, and Y is the total number of leaves on each plant. The mean number of lesions per leaf (LESNO) was calculated for each plant with the following equation:

[14] $$\text{LESNO} = Z/10$$

where Z is the total number of lesions counted on the sample leaves, and 10 is the number of sample leaves. Using Statistix (Analytical Software, Tallahassee, FL), a paired t test was performed to see if each leaf wetness and temperature interaction produced a significant difference in the LESNO on the top leaves compared with the bottom leaves on each of the plants. It was determined that there was a significant difference in the susceptibility of young and old leaves if $P \leq 0.05$.

An F test was performed to determine if combining the data from both experiments was warranted (Bulger et al. 1987). The relationship between PINFL and LESNO was assessed using Pearson's correlation coefficient analysis. In the absence of leaf wetness (treatment with LW 0), few disease symptoms were observed in the second repetition of the study, resulting in disease severity data that were not normally distributed. Therefore, data were log transformed prior to analysis of variance. The relationship between leaf wetness duration and temperature for infection by *E. mespili* was described following the analysis of variance and multiple-regression procedures. Linear, quadratic, and cubic effects of leaf wetness duration and temperature and their interactions on PINFL were tested. Based on SAS (SAS Institute Inc., Cary, North Carolina) stepwise regression, parameters not significantly different from zero ($P \leq 0.05$) were omitted from the equation, using backward elimination, unless higher-degree terms of the corresponding variable were associated with significant parameters. This stepwise procedure was conducted until a simple model with the best distribution of parameter estimates was derived. Using the simplified regression model, the coefficient of determination (R^2), and the pattern and distribution of residuals were examined for each repetition. A coefficient of determination was also calculated for both repetitions combined.

3.3.2. Results

When the temperature treatments were combined for each leaf wetness period, both the PINFL and LESNO increased with an increase in the leaf wetness period (Fig. 3.3). In general, disease severity trends relative to increasing leaf wetness periods were similar in both repetitions of the study, despite the fact that the first repetition did not have as many treatments as the second, and PINFL and LESNO values were both higher in the second repetition. In the second repetition a small number of lesions formed without a period of leaf wetness when the plants were exposed to temperatures of 20 or 30°C after inoculation.

An F test indicated that the results of the two studies were not significantly different from each other ($P \leq 0.05$). Therefore, the data from the two repetitions were pooled. Symptoms were not observed on plants which were not inoculated. Increasing the leaf wetness period after the plants were inoculated increased the PINFL and LESNO for all temperature treatments (Fig. 3.4). Considerably more infected leaves and lesions per leaf developed at temperatures between 10 and 20°C, and when the 18°C leaf wetness periods were greater or equal to 12 h. There was a significant correlation ($r = 0.78$) between combined data of PINFL and LESNO, so only PINFL was used for regression analysis. Percent-infected leaves per plant provided an effective measure of disease development over the entire plant. The relationship between the 18°C leaf wetness durations and subsequent temperature in regards to PINFL is as follows:
[15] $\text{Log}_{10}(Y+2) = -b_0 + b_1 T - b_2 T^2 + b_3 T^3 + b_4 W - b_5 W^2 - b_6 T W + b_7 T^2 W - b_8 T^3 W$
where Y is the predicted PINFL, T is temperature, and W is leaf wetness duration. The significant parameter estimates for equation [15] are listed in Table 3.5. The R^2 values were 0.94 and 0.85 during the first and second repetitions, respectively, and a random pattern of residuals was observed across the range of predicted means. When the repetitions were combined, the R^2 value was 0.79.

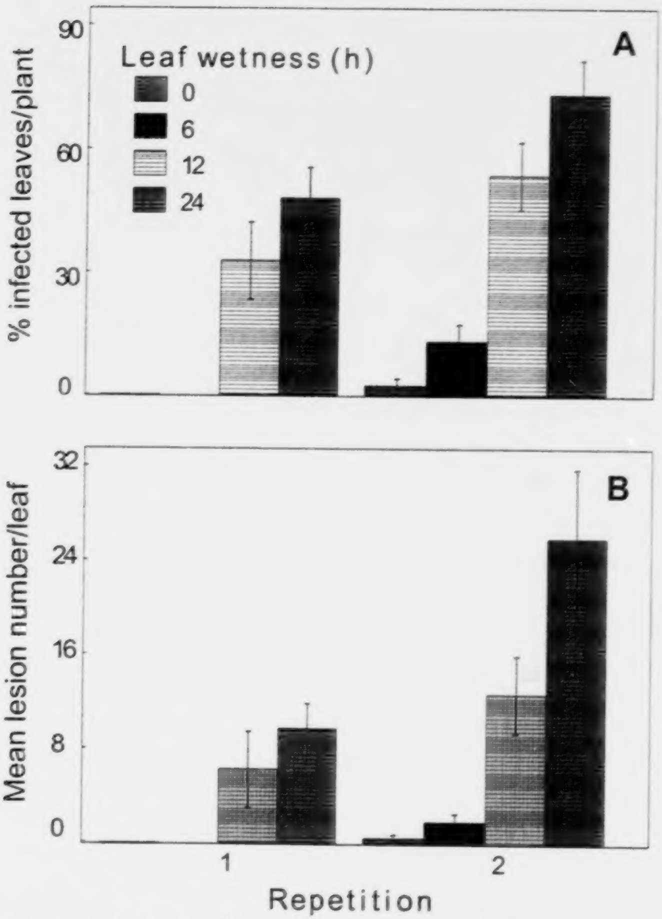

Figure 3.3 *Entomosporium mespili* development on saskatoon plants relative to leaf wetness (LW) duration (0, 6, 12, 24 h) for combined temperature treatments from two repetitions of a growth-room study. **A.** The mean (± standard error) percent infected leaves per plant (PINFL). **B.** The total mean (± standard error) lesion number per leaf (LESNO). No disease development was observed in the first repetition when the leaf wetness duration was 0 h. There was no treatment for 6 h of leaf wetness in the first repetition of this study; therefore, the space left for this column is blank.

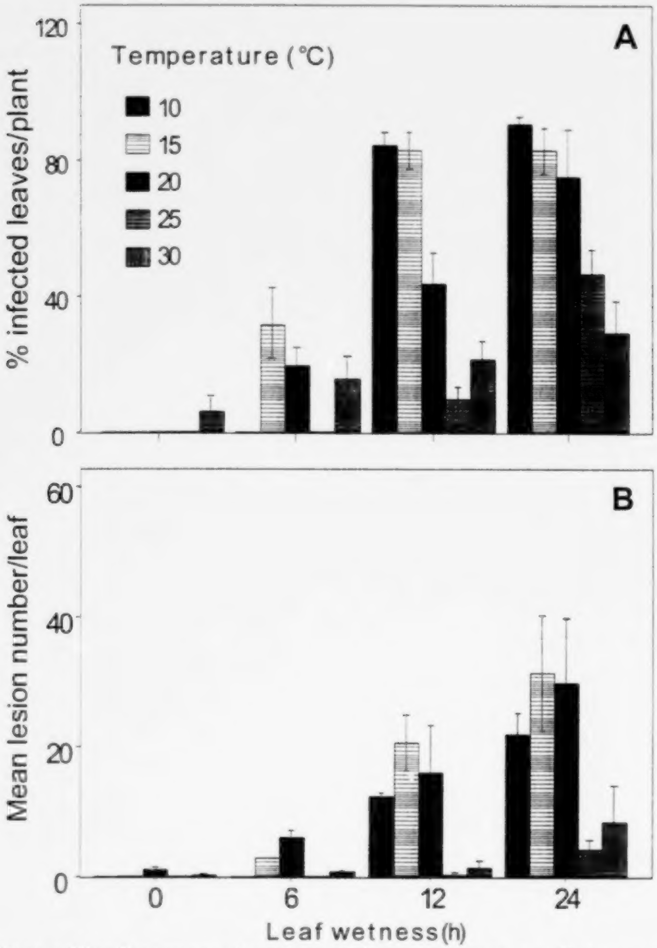

Figure 3.4 Effect of leaf wetness duration and temperature on *Entomosporium mespili* development on saskatoon plants. **A.** The mean (± standard error) percent infected leaves per plant (PINFL). **B.** Total mean (± standard error) lesion number per leaf (LESNO).

Table 3.5 Significant parameter estimates and their standard errors used in empirical model [15] to forecast the effects of leaf wetness duration and temperature on the development of *Entomosporium mespili* on saskatoon leaves.

Parameter	Estimate	Standard Error
b_0	-23.3187	5.6821
b_1	4.0839	0.9793
b_2	-0.2190	0.0520
b_3	0.0037	0.0009
b_4	1.3094	0.4158
b_5	-0.0082	0.0019
b_6	-0.1625	0.0712
b_7	0.0089	0.0038
b_8	-0.0002	0.0001

Younger leaves closest to the top of the plant were generally more susceptible than older leaves found near the bottom of the plant for each leaf wetness and temperature treatment (Table 3.6). At 15°C, LESNO differences were significant at 6, 12 and 24 h of leaf wetness. As the leaf wetness period increased, so did the temperature range at which there was a significant difference in the LESNO on the top versus bottom leaves.

3.3.3. Discussion

This study demonstrated that the duration of leaf wetness when fixed at a constant temperature of 18°C is an important factor for ELBS disease development on saskatoon. There was a large increase in disease development with duration of leaf wetness of 24 h. However, as our study did not look at leaf wetness periods beyond 24 h, it was not possible to determine if 24 h was the optimal leaf wetness period for infection of saskatoon. Similarly, studies of other pathogens (Grove 2002; Monroe et al. 1997) and *E. mespili* on *Photinia* X *fraseri* (Baudoin 1986a) also revealed a pattern of increased disease with increasing leaf wetness. In these studies, a period of 24 h was the longest duration examined. The critical temperatures required for *E. mespili* conidia germination during the leaf wetness period was not considered in this study; however they should be examined more closely in a future study.

Table 3.6 Effect of leaf wetness duration and temperature on the average number of lesions per leaf caused by *Entomosporium mespili* on saskatoon.

	Leaf wetness duration (h)							
	0		6		12		24	
Temperature (°C)	Top	Bottom	Top	Bottom	Top	Bottom	Top	Bottom
10	0	0	0	0	20.6*	4.1	33.9*	9.6
15	0	0	4.7*	0.9	23.5*	17.5	46.3*	16.2
20	0	0	2.1	0.3	18.4*	2.3	33.9*	12.0
25	0	0	0	0	0.3	0.2	6.1*	2.1
30	0.4	0.2	0.8	0.5	2.0	0.6	11.8	4.8

Note: Samples consisted of five leaves from the top portion (young leaves) and five leaves from the bottom portion (older leaves) of each plant.

* Data followed by an asterisk indicate a significant difference between the average number of lesions per leaf on the top versus bottom leaves for individual treatment combinations.

Along with leaf wetness, temperature also had an important impact on disease development. Temperature influences both pathogen development and plant growth (Harrison 1992). In the growth-room study, saskatoon stem basal diameter grew minimally at 10ºC and 30ºC, and optimally at 20ºC (data not shown). For pathogen growth and development, temperatures above 20ºC suppressed *E. mespili* lesion development on saskatoon, while maximum infection of saskatoon by *E. mespili* occurred between 10 and 20ºC after 24 h of leaf wetness at 18ºC post inoculation. Baudoin (1986a) showed that the optimum temperature for *E. mespili* development on *Photinia* X *fraseri* was 20ºC; however, infection was only slightly less at 15 and 25ºC. In contrast, the present study showed that infection was reduced on saskatoon plants when the temperature was 25ºC or higher. Baudoin (1986a), Horie and Kobayashi (1979) and Rosenberger (1981) indicated that 20 to 25ºC is the optimal temperature range for *E. mespili* conidia to develop on different host plants. Besides the effect of temperature on disease development, some preliminary data from the growth-room experiments suggests that the incubation period decreases with increasing temperature up to 20ºC and then stays approximately constant (data not shown). Such a response could potentially have the effect of reducing the duration of each infection cycle, and ultimately reduce the time required for an epidemic to develop (Fry 1982a). Further work is needed with larger sample sizes to confirm the relationship of leaf wetness duration and temperature with the incubation period required for disease development.

Leaf wetness is not only important for disease development, but its duration will also influence the minimum temperature required for disease to develop. Although our study showed that a leaf wetness period is important for disease development, a few lesions developed without leaf wetness at temperatures of 20ºC and 30ºC in the second repetition of the study. This could have resulted from the leaf stomata being open more frequently when the temperature was 20 or 30ºC. Although conidia of *E. mespili* typically germinate and then form an appressorium that directly penetrates the leaf cuticle, van der Zwet and

Stroo (1985) showed that *E. maculatum* Lév., which is a synonym for the anomorphic state of *E. mespili*, can enter and infect pear leaves through their stomata. This may explain why disease symptoms increased when the temperature was raised to 30°C for an extended period.

The present study suggests that the relationship between an 18°C leaf wetness duration and subsequent temperature treatment can be used to predict the likelihood of disease symptom development. Similar relationships between leaf wetness and temperature have been well documented for a variety of plants and diseases (Baudoin 1986a; Evans et al. 1992; Thompson and Jenkins 1985). Although the regression equation developed here applies to the controlled environmental conditions of the present study, it lays the foundation for the development of a model for fungicide spray scheduling under field conditions. When used in combination with a fungicide application program in the field, this regression equation has the potential to help reduce early season leaf infection. A reduction in the amount of diseased tissue on plants has the potential to limit inoculum abundance for saskatoon fruit infection. Nevertheless, it is still conceivable that fruit may become infected directly from inoculum that is released from overwintering structures that can be found on young branch tissue, dead infected leaves, and possibly on infected pedicels and fruit that remain on or around the plant (Horie and Kobayashi 1980b; Stathis and Plakidas 1959).

Tissue susceptibility to a particular pathogen can vary with organ age. In our experiment, young newly expanded saskatoon leaves near the top of the plant tended to develop more lesions per leaf on average than older, more physiologically mature leaves on the lower half of the plant. This distinction was particularly evident when leaf wetness durations and temperature were optimal. Ronald et al. (2001) found that young saskatoon leaves were more susceptible to infection than older leaves. Baudoin (1986b) also showed that *E. mespili* infection was more aggressive on young leaves of *Photinia* X *fraseri* than on older leaves. Jacobs et al. (1996) showed that young leaves of *Photinia* species could be infected by *E. mespili* 10 to 30-fold more often than mature leaves.

Understanding how the chemical and/or physical condition of a leaf changes as it matures provides insight into how older leaves become more resistant to symptom development. For example, Wetzstein and Sparks (1983) noted that higher levels of resistance in older leaves of *Carya illinoensis* Wang. were associated with low trichome density, high phenolic content in the mesophyll, and thick cuticles. As a plant leaf begins to age, waxes are built up on their surface. Waxes on leaf and fruit surfaces form a water-repellent layer and thereby prevent the formation of a film of water in which pathogens might be deposited and germinate or multiply (Agrios 1997). *Entomosporium mespili* primarily infects plants like *Photinia* through direct cuticle penetration, and therefore a thicker cuticle may provide superior physical protection from infection.

In conclusion, this study created a regression equation that defines disease development based on the interaction between leaf wetness duration and subsequent temperature treatment. To improve the functionality of this model for predicting disease development in a field situation a future study should be conducted to model the critical temperatures required for *E. mespili* conidia germination during the leaf wetness period. It was also shown that susceptibility of young and old leaves will depend on the leaf wetness duration and temperature. Ultimately, the empirical model developed in this study will be incorporated with a measurement of inoculum production, time of conidia release, and host susceptibility to create a mechanistic model for describing *E. mespili* development on saskatoon plants.

3.4. Estimating Inoculum Production Relative To Percent Leaf Area Infected And Environmental Conditions Required For Stimulating Release Of E*ntomosporium mespili* Conidia

Polycyclic diseases, like ELBS, can be efficiently suppressed by reducing initial inoculum and/or by limiting potentially rapid rates of inoculum increase (Fry 1982b). Thompson and Jenkins (1985) showed that a reduction in lesion area results in a lower number of *Colletotrichum lagenarium* (Pass.) conidia produced from infected leaves of *Cucumis sativus* L. Models that integrate leaf wetness duration and temperature to forecast disease development (Grove 2002; Monroe et al. 1997; Tamm and Flückiger 1993) can be integrated with a fungicide spray program to help reduce disease. A similar disease-forecasting model based on leaf wetness duration and temperature for *E. mespili* on saskatoon was developed in Chapter 4. The accuracy of the above forecasting models would be improved if information on inoculum production and inoculum release were included. There are many studies that have examined the epidemiology of fungal spore release in relation to environmental factors (Fernando et al. 1997; Paulitz 1996; Pinkerton et al. 1998). The objectives of this study were: 1) to relate the percent of saskatoon leaf area infected to the relative concentration of *E. mespili* conidia released from the infected tissue and develop a regression equation for this relationship; 2) to identify when *E. mespili* conidia are released in relation to prevailing environmental conditions during the growing season; and 3) to determine if the amount of conidia released is related to the amount of rainfall during precipitation events.

3.4.1. Materials and Methods

Three ‘Smoky’ saskatoon orchards were used as a source of leaf samples required for this study. Sampling was done over a two-year period (1999 and 2000). The first orchard (Carman Site 1) was located near Carman, Manitoba, and was not treated for control of *E. mespili* in either study year. A second orchard (Carman Site 2) was located adjacent to Carman Site 1 and received fungicide treatments as determined by the grower during the 1999-growing season, although the treatments were relatively ineffective for controlling the disease. The third site, which also did not receive any fungicide treatments, was located at the University of Manitoba, Winnipeg, Manitoba. Data from this site were collected during the 2000 season.

Leaf samples from each orchard were collected once per week between early June and the end of August. Five leaves per plant were randomly collected from each of 10 plants in each orchard during each sampling period. The five leaves from each plant were placed on a piece of blue construction paper and photographed using a digital camera.

The mean percent leaf area infected (PLAI) of the samples was then determined by importing the pictures into the software program Assess for Windows (APS Press, Saint Paul, MN). Digital image analysis programs have been used to quantify disease areas in several pathosystems (Blanchette 1982; Chungu et al. 1997; Lindow and Webb 1983; Martin et al. 1999; Niemira et al. 1999; Tucker and Chakraborty 1997). Assess for Windows was selected over other programs because it did not require specialized equipment or software application expertise to detect diseased areas on infected plant tissue. The program first identifies the total leaf area and then assesses the PLAI using pixel color analysis.

Following PLAI determination, each sample of five leaves was placed in a beaker with 10 mL of water and several small glass beads to help stimulate conidia release during a period of agitation. The beakers and their contents were agitated using a flat-top oscillator for 1 h. A preliminary study determined that 1 h was adequate to induce maximal conidia release from infected leaf tissue. Following agitation, an aliquot was extracted using a pipette and the conidial suspension was quantified using a hemacytometer. The relative concentration of conidia (RCC) in each sample was determined by averaging the number of conidia observed from six 1 mm^2 grids on the hemacytometer.

The software program Statistix for Windows (Analytical Software, Tallahassee, FL) was used to analyze the RCC and PLAI data. For each sample site and year, the RCC to PLAI was compared using Pearson's correlation coefficients. An *F* test was used to determine if combining the data means from each sampling site and year was justified (Grove et al. 1985) and a combined analysis was conducted where appropriate. The interaction between the combined RCC and PLAI data was illustrated using scatter plot and regression analyses.

An Adcon Telemetry (Adcon Telemetry, Klosterneuburg, Austria) weather monitoring station was used at the Winnipeg orchard to quantify rainfall during the study. These weather stations, which are maintained by the Agrometeorological Center of Excellence (ACE) in Manitoba, provide environmental variable monitoring with real-time and site specific microclimate measurements.

During the 1999 field season, Vaseline™-coated microscope slides were used to detect conidia release. Several different saskatoon plants with a history of ELBS disease were selected for this study at the beginning of June in the Winnipeg orchard. Two Vaseline™-coated microscope slides were hung parallel to and at a height of 1 m from the ground from randomly selected branches just prior to rainfall events. After rainfall ceased, the slides were collected for analysis. A light microscope (40X magnification) was used to locate and identify conidia on the microscope slides.

A trap designed to capture splash-dispersed conidia (see Appendix A) was placed in the Winnipeg orchard on Julian day 182 in 2001 and on Julian day 121 in 2002. In both years, conidia were trapped until Julian day 243. The trap was placed in the periphery of the saskatoon plant canopy in an attempt to ensure the greatest capture of runoff rainwater from leaves (Fig. 3.5). In a saskatoon plant that measured 3.5 m in height, the trap was placed 1 m from the ground and 30 cm in from the outer edge of the canopy. The trap rotated the position of a test tube each hour using a battery powered electric step motor. After each precipitation event, all of the test tubes containing rainwater were retrieved and the number of conidia per mL of rainwater was estimated for each hourly sample using a hemacytometer.

Monitoring of disease symptoms began when 50% of the overwintering buds in the Winnipeg orchard reached the stage of bud break. The phenological stage of flowering was defined as the time when an estimated 50% of the inflorescences in the orchard had one open flower. A weekly estimate of PLAI, as described above, was determined between early July and late August by randomly selecting 5 leaves from each of 10 randomly selected plants soon after disease symptoms in the orchard were first observed.

Statistix for Windows was used to perform a Pearson's correlation analysis to determine if the number of conidia per mL of trapped rainwater was dependent of the total amount of rainfall during a precipitation event. For both study years a regression curve of PLAI in the orchard relative to day of the year was plotted and visually compared to rainfall and trapped conidia data. Conidia trapped in the hours following the start of rainfall events were considered. This was accomplished by assessing conidial presence in 1 h increments after the start of each rainfall event.

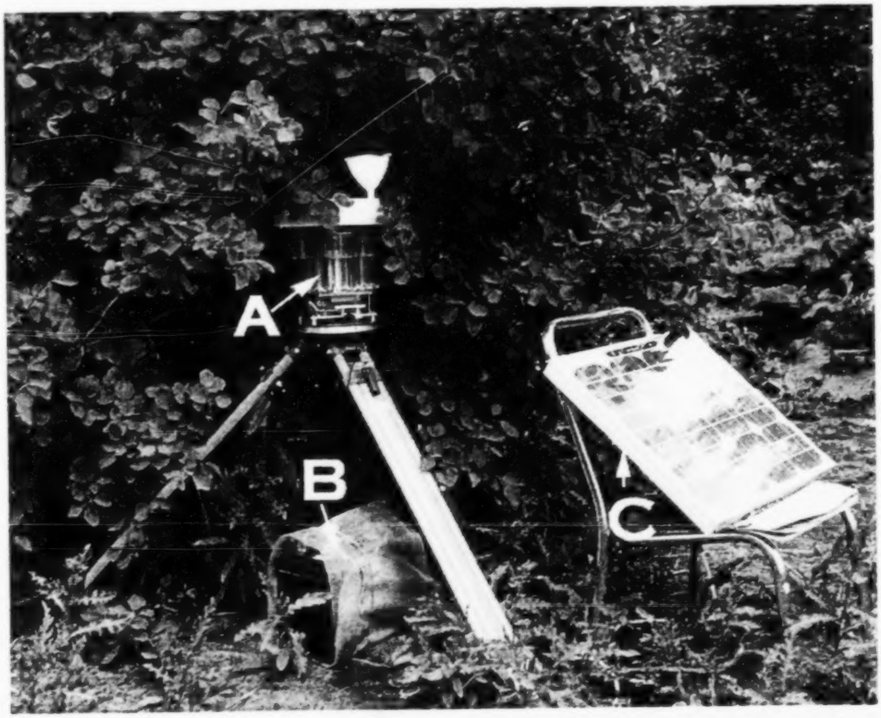

Figure 3.5 The splash-dispersed conidia trap positioned 1 m from the ground in the periphery of a saskatoon plant canopy (**A**). Power for the electrical step motor in the trap was supplied by a 12-V deep cycle battery (**B**), which was recharged by a 30-Watt solar panel (**C**).

3.4.2. Results

When the RCC was compared against PLAI the Pearson's correlation coefficients (r) were 0.73 and 0.83 for 1999 and 2000, respectively, at Carman Site 1, 0.72 at Carman Site 2 in 1999, and 0.69 at Winnipeg in 2000. An F test showed that the results from each study site and year were not significantly different ($P \leq 0.05$), and so the data were combined for further analysis. Regression equation [16] best describes the positive linear relationship between the percent of saskatoon leaf area infected to the relative concentration of *E. mespili* conidia (Fig. 3.6).

$$Y = 5.34\text{x}10^{-2} * X \qquad [16]$$

where Y is the RCC and X represents PLAI. The coefficient of determination (R^2) for this equation was 0.83.

Flowering began on Julian day 133 in 2001 and day 150 in 2002. Between flowering and Julian day 243 there were 36 rainfall events in 2001 for a total of 293 mm and 38 events in 2002 for a total of 328 mm (Fig. 3.7A and 3.8A). Despite the fact that no conidia were trapped at the beginning of the flowering period, the first disease symptoms appeared 5 d after the first rainfall event that occurred 1 d or more after the date of flowering. This observation was agreeable with data from several other locations and years (Appendix B), and suggests that initial conidia release could have occurred as early as Julian day 135 in 2001 and Julian day 156 in 2002 at the Winnipeg Site based on a 5 d incubation period. Conidia were first trapped on Julian day 184, and continued to be trapped until Julian day 243 in both 2001 and 2002 (Fig. 3.7B and 3.8B). The correlation between the number of conidia per mL trapped and the amount of rainwater collected during each precipitation event was not significant in 2001 ($P \leq 0.05$), but was in 2002 ($r = 0.74$). Considerably fewer conidia were trapped during rainfall events in 2001 than in 2002. Conidia were trapped primarily in the first 2 h of a rainfall event. There were only two rainfall events, both in 2002, which caused conidia to be released for several hours beyond the first 2 h. These two events yielded the highest number of conidia during the entire season.

For each year of the study, conidia were trapped only during rainfall events that occurred after the PLAI began to increase exponentially (Fig. 3.7C and 3.8C). In general, the relationship between PLAI and Julian day was described by equations [17] and [18], in 2001 and 2002, respectively:

$$Y = 1.14\text{x}10^{-1}\ e^{0.0536((-132) + X)} \qquad [17]$$

$$Y = 1.72\text{x}10^{-2}\ e^{0.0696((-132) + X)} \qquad [18]$$

where Y is PLAI and X represents Julian day.

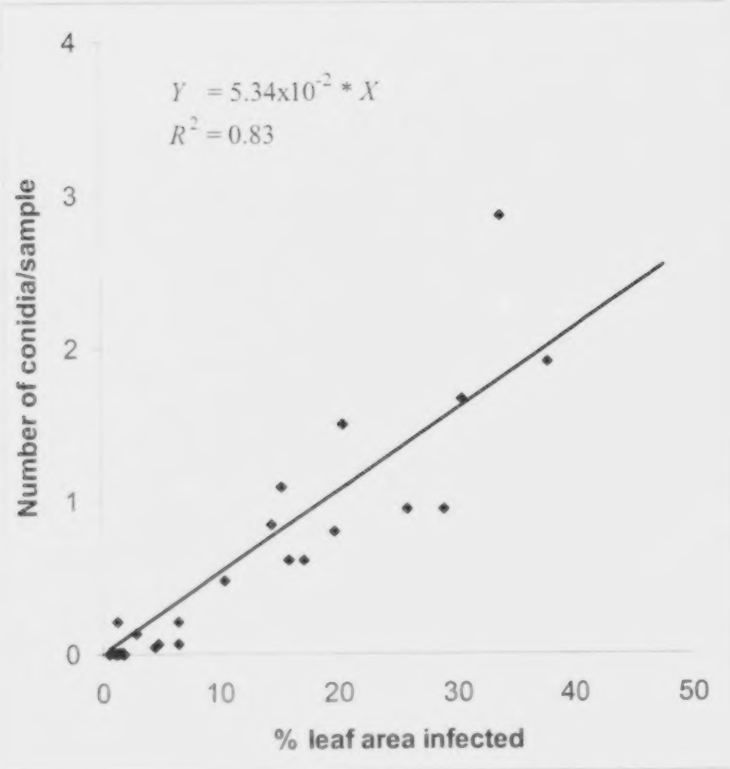

Figure 3.6 Regression analysis between the percent of saskatoon leaf area infected and the relative concentration of *Entomosporium mespili* conidia. Data were collected at Winnipeg and Carman Sites 1 and 2 in 1999 and 2000.

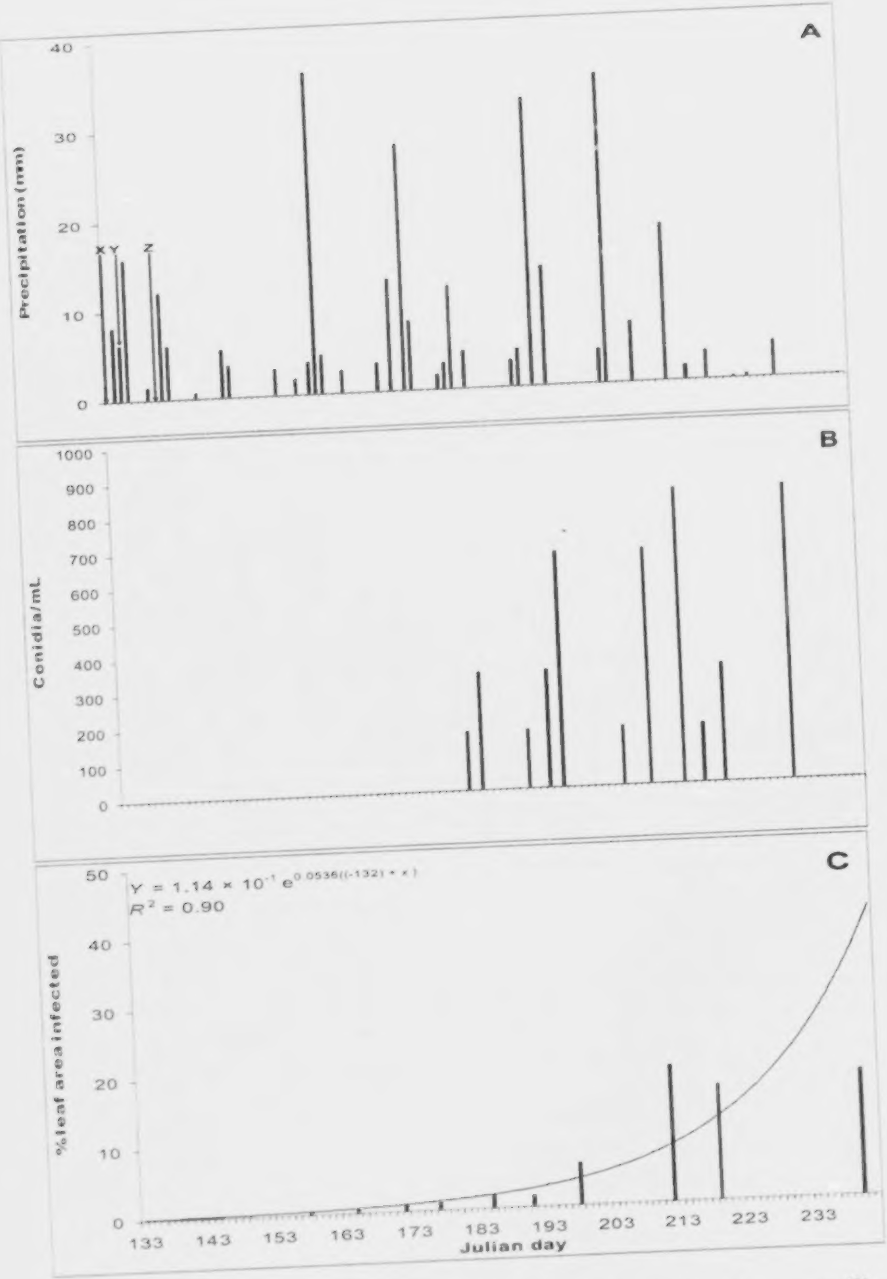

Figure 3.7 Precipitation (A), numbers of conidia of Entomosporium mespili per mL of rainwater collected (B), and mean percent leaf area infected (C) in the Winnipeg orchard in 2001.

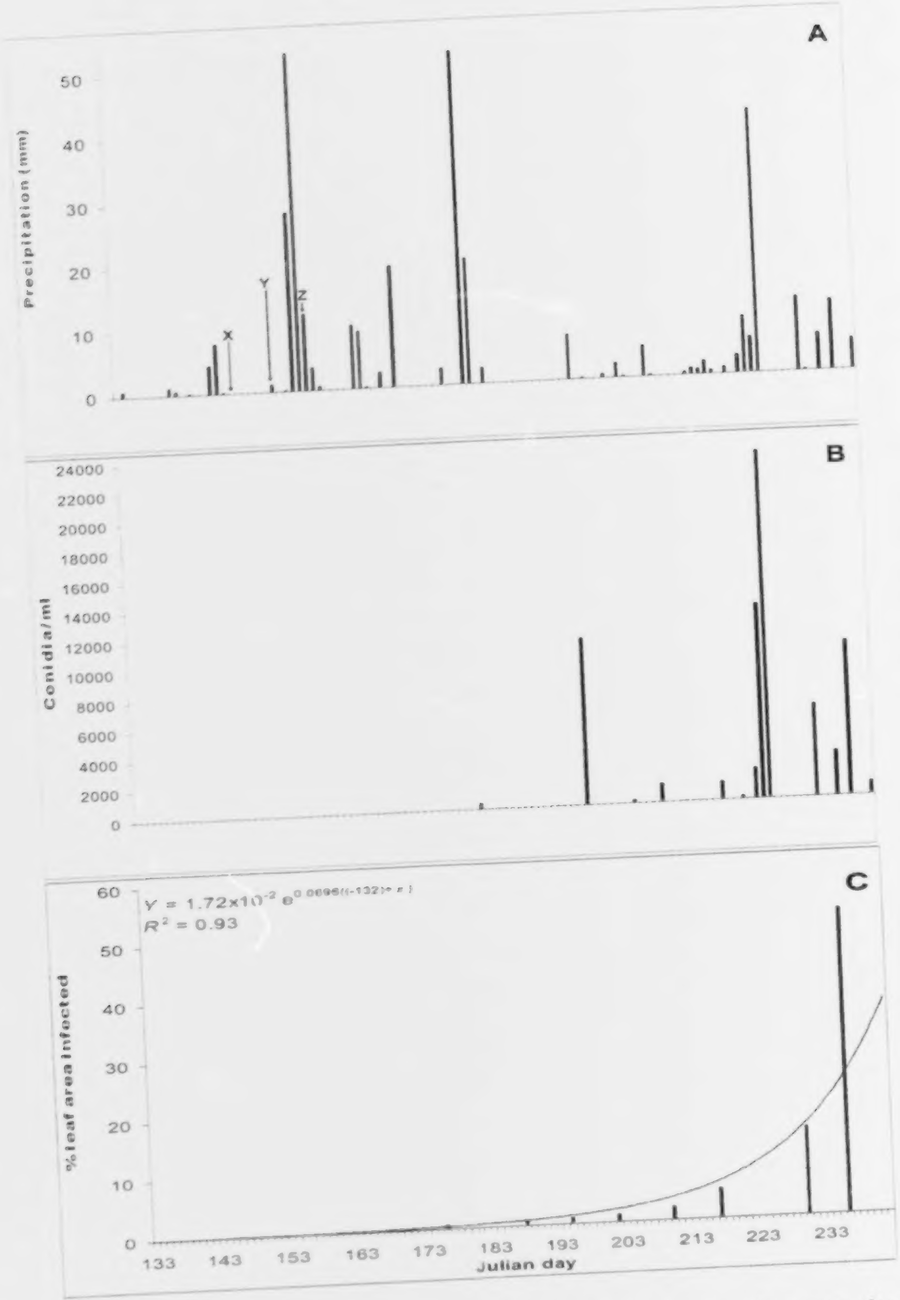

Figure 3.8. Precipitation (**A**), numbers of conidia of *Entomosporium mespili* per mL of rainwater collected (**B**), and mean percent leaf area infected (**C**) in the Winnipeg orchard in 2002.

3.4.3 Discussion

Combinations of leaf wetness duration and temperature that favor ELBS disease development on saskatoon leaves were identified in Section 3.3. However, despite the presence of ideal weather conditions for disease development, it would be of little value forecasting a possible disease outbreak if inoculum was not present. Inoculum potential is an important epidemiological factor that can be used to predict disease epidemics for both monocyclic and polycyclic pathogens (Fry 1982a; Specht and Griffin 1988; Yitbarek et al. 1988).

Results from the inoculum production study indicated that the relative concentration of *E. mespili* conidia is strongly correlated with the percent of saskatoon leaf area infected. Therefore, an assessment of PLAI at the end of the growing season could provide an estimate of the inoculum overwintering in an orchard. Estimates of inoculum levels going into the winter have been valuable for implementing an early spring fungicide spray program to control pathogens, like *Venturia inaequalis* (Cooke) G. Winter, on apple trees (Boone 1971). This same system for disease control would benefit saskatoon growers because *E. mespili*, like *V. inaequalis*, infects young saskatoon leaves in early spring when they are most susceptible to infection (Ronald et al. 2001).

Knowledge of inoculum potential within an orchard is an important part of understanding the epidemiology of a pathogen; however, it is also important to know when conidia dissemination occurs relative to prevailing environmental conditions. An array of factors affecting the release of fungal spores have been identified, including temperature (MacHardy and Gadoury 1986), light (Luley and McNabb 1989), vibration (Leach 1980), humidity (Gadoury et al. 1998), free water (Stensvand et al. 1998), and precipitation (Madden et al. 1996). In a preliminary study, a Burkard 7-day volumetric spore trap (Burkard Manufacturing Co. Ltd., Hertfordshire, England) was used throughout a growing season, which had severe disease, to confirm that air currents do not disseminate conidia of *E. mespili* (data not shown). Ascospores may be vectored by wind in the early part of the season, although no study, including this one, has conclusively shown this phenomenon to occur in saskatoon orchards. More research is required in this area. Results from both the Vaseline™-coated microscope slides and the splash dispersed conidia trap did show that *E. mespili* conidia are dispersed by rain-splash during precipitation events after disease symptoms have already been observed in the orchard. Madden et al. (1996) found that as rain intensity increased so did the total number of *Colletotrichum acutatum* Simmonds conidia in rain splash droplets. It could also be expected that more inoculum will be trapped as the total amount of rainfall increases. This was observed in the second year of the present study. The lack of a correlation between the number of conidia trapped and the amount of rainfall in the first year of this study could be explained by examining the interval of time between rainfall events that caused conidia to be disseminated. For instance, there was a long period in the middle of the growing season (Julian day 188 to 199) that did not have a rainfall event in

2002, whereas rainfall occurred no more than 6 d apart in the middle of season 2001. The increased time between rainfall events allows acervuli to mature and thus more mature conidia could potentially be released from previously infected plant tissue during future rainfall events. van der Zwet and Stroo (1985) indicated that it takes 2 to 4 wk from the time of infection for mature conidia of *E. maculatum* Lév. to exude from acervuli on pear leaves. Thus, a quantitative relationship between the time of rain events and the numbers of conidia trapped is not unexpected because of the dynamics of the conidia production process. It was also observed in our study that a dry period of two or more hours between rainfall events is required to stimulate conidia release. This dry period may help to desiccate the surface of mature acervuli. Rainwater would then be able to wash conidia from the surface of the exposed acervuli (Ronald and St-Pierre 2002).

Although no conidia were trapped around the time of flowering, the first disease symptoms appeared 5 d after the first rain event that occurred 1 d or more after flowering in both years in this study. Disease symptoms first appeared 43 and 23 d before conidia were first trapped in 2001 and 2002, respectively. It is possible that conidia levels were not high enough to be trapped by the single splash-dispersed conidia trap at this time. Future work is required to identify the earliest date that *E. mespili* conidia can be released during the growing season. To do this, it is suggested that conidia trapping should begin before flowering and that several splash-dispersed traps and multiple Vaseline™-coated microscope slides should be used. One possible explanation as to why no disease symptoms developed prior to the first rain event that occurred 1 d or more after the phenological date of flowering is that no susceptible leaf surface has developed and water is shed by gravity from the unopened leaves. Moreover, it is possible that conidia are released during these early season rain events but have poor germination due to low temperatures at this time of year. This may result in little or no disease symptom development despite the presence of conidia.

During the course of the study, *E. mespili* conidia were primarily released during the first 2 h period of rainfall events with only 2 instances where conidia were trapped beyond the first 2 h. One possible explanation for the short period of conidia release is that the conidia may have been washed downward in the canopy past the point of collection on the trap during the first 2 h of rainfall. To verify that conidia are only disseminated in the first 2 h of rainfall events, the height of the trap could be lowered in future studies to allow more infected plant canopy to hang over the collection point on the trap. The placement of the trap may have also influenced why fewer conidia were trapped in the 2001 season. It is possible that by chance, less infected material was located above the collection point on the trap during 2001. Another possible reason for limited conidia movement beyond the first 2 h of a rainfall event may be associated with the appendages on the conidium. Mims et al. (2000) suggested that these appendages may help to attach the conidium to the leaf surface preventing their movement far beyond their source of dissemination.

The exponential start of PLAI increase was associated with the first time that conidia were trapped. The exponential equations created in this study adequately described the increase in PLAI throughout most of each season. However, despite the fact that the R^2

values were quite high, the accuracy of both models decreased after Julian day 228. Disease development at the end of the season appeared to correspond with the number of rainfall events that occurred during this period. In the drier late-season period in 2001, actual PLAI was less than the predicted amount. This may have been the result of fewer conidia release events. In contrast, the amount of PLAI in 2002 was greater than the predicted PLAI and this may be the result of more conidia release events during that period. These results suggest the importance of a future study to investigate the timing of rain events relative to disease development throughout the growing season.

In summary, it was shown that the percent of saskatoon leaf area infected can be used to estimate the production of *E. mespili* conidia in an orchard using regression analysis. This relationship may be used to provide an estimate of overwintering inoculum, which will be available to initiate disease in the spring. A splash-dispersed conidia trap was used to show that *E. mespili* conidia are released primarily during the first 2 h period of rainfall events, and that the number of conidia per mL of rainwater collected is not entirely dependent upon the total amount of rainfall during a precipitation event. Saskatoon plants initially become susceptible to conidia that are disseminated by rainfall events occurring 1 d or more after the phenological event of flowering. However, more research is required to determine if ascospores play a role in initial infection. Conidia were initially trapped on Julian day 184, which coincided with the start of exponential PLAI increase, and continued to be trapped until Julian day 243. Multiple late season rain events can promote disease level increase by stimulating conidia release.

3.5. The Development Of A Dynamic Disease-forecasting Model To Control *Entomosporium mespili* On *Amelanchier alnifolia*

There are three fungicides registered to control ELBS on saskatoon plants in Canada: Funginex (triforine), Topas 250E (propiconazole), and Kumulus (sulfur) (Bains 2000). The best disease control is attained using spray applications of Topas 250E at the white tip, petal drop, and green fruit stages as recommended by Lange et al. (1998) and St-Pierre (1997). Once applied, Topas 250E is registered to effectively control most diseases for a period of up to 21 d. Therefore, current spray recommendations promote overuse of the fungicide given anthesis in saskatoon plants lasts 3.5 ±0.8 d (McKay 1973; St-Pierre and Steeves 1990), with the white tip and green fruit stages occurring shortly before and after this period, respectively. Furthermore, this fungicide application program assumes that weather conditions required for disease development are present during this period. Overuse of fungicides can be damaging to the environment, consumer health, and grower profits (Babcock et al. 1992). One way to improve disease prediction is to develop a disease-forecast model based on environmental conditions conducive to disease development. Such a disease-forecasting model would help to ensure optimal use of Topas 250E, and therefore prevent overuse of the product.

The justification for constructing a disease-forecasting model can be based on economic, environmental, and political factors (Royle 1985), and its aim is to reduce uncertainty about control decisions by providing a quantitative description of disease pressure (Tait 1987). Although a disease-forecast model could be either empirical or mechanistic (Zadoks 1984), most of the disease-forecast models created thus far are empirical. An example of such an empirical model is the non-linear regression equation that was used to predict the infection of cherry and peach foliage by *Wilsonomyces carpophilus* (Lev.) when various leaf wetness durations and temperatures were introduced after inoculation (Grove 2002). Microclimate variables have comprised the backbone of many disease-forecast models (Grove et al. 1985; Mathieu and Kushalappa 1993). However, once a greater understanding of the biological system required for disease development is attained then a mechanistic, or rather a dynamic model for disease development can be created. Integrating the components of the disease triangle, which includes the pathogen, the host, and their environment, will create a disease-forecast model that is comprised of a more complete biological system (Agrios 1997). Mechanistic models attempt to explicitly represent causality between several variables (Whisler et al. 1986). As a result, several regression equations will often be used to describe the interaction between the pathogen and each component of its environment, much like the neural network model for tan spot and stagonospora blotch in wheat (De Wolf and Francl 2000).

Once a disease-forecasting model has been constructed, its predictive ability must be assessed. Disease-forecast models are typically validated by comparing observed disease symptom development from lab and or field evaluation trials against predicted values that have been generated by a regression model. Asher and Williams (1991) assessed a model for sugar-beet powdery mildew based on weather data in this fashion. Bruhn and Fry (1981) evaluated model performance by comparing real disease progress curves in four potato cultivars with disease progress curves generated by a model based on weather data.

The objectives of this study were: 1) to create a dynamic disease-forecasting model for estimating ELBS disease in saskatoon orchards based on the epidemiological and phenological information described in Chapters 3 to 5; 2) to incorporate information on host susceptibility relative to leaf age; 3) to test the accuracy of the disease pressure index equation used in the disease-forecasting model against observed disease development in two saskatoon orchards over several years; and 4) to expand the model to include control mechanisms and to test its effectiveness.

There were two main sources of information used in the development of the disease-forecast model for ELBS on saskatoons. First, information was obtained regarding phenological development of saskatoon, the relationship of disease to leaf wetness duration and temperature, inoculum production, and inoculum release. Secondly, in addition to these data, specific data were obtained on the relationship of disease development relative to specific weather variables at the Winnipeg and Carman orchards between 1999 and 2002. Both orchards contained mature 'Smoky' saskatoons that had a history of severe ELBS disease. Using the Adcon Telemetry (Adcon Telemetry, Klosterneuburg, Austria) weather stations established in each orchard in 1999, precipitation above the saskatoon plant canopy and leaf wetness and temperature at 1 m in height and 15 cm within the outer edge of the canopy were measured. There were also some parameter assumptions built into the model. The various model components will be elaborated upon in the context of the description of model development.

3.5.1 Phenological Development of Saskatoon

The phenological stage of bud break in each orchard was determined visually according to section 3.2. The date of bud break was used to start the phenological component of the disease-forecasting model (Fig. 3.9). Phenological development of saskatoon from the date of bud break until flowering and fruit harvest was predicted using SASK-Day heat units and average historical daily heat unit data that were collected between the years 1999 and 2001 at the Winnipeg and Carman orchards. The SASK-Day model was used to calculate the sum of heat units accumulated from the time of bud break and then from this point historical daily heat unit data were used to predict the 112.1 and 515.9 heat unit sums that are required to complete the phenological stages of flowering and fruit harvest, respectively. A hypothetical prediction of flowering from three different times is illustrated in Figure 3.10. In this example, the accuracy of the system increased as more time was allowed to accumulate actual heat unit data before the prediction was made. The earliest possible date that was considered successful for predicting a future event was identified as the time when the actual dates of flowering and fruit harvest fell within 2 d of their respective predicted dates.

Once the phenological stage of bud break is reached the disease-forecasting model waits for the first rain event that occurs 1 d or more after the date of flowering, because this was considered the beginning of the susceptible period. The date of flowering can be predicted using the phenological model described above, or entered directly into the disease-forecasting model when more than 50% of the inflorescences in the orchard have reached the flowering stage of development. The phenological stage of flowering was defined in section 3.1 as the time that the petals of the apical flower in the inflorescence were completely unfolded to expose the reproductive structures of the flower.

3.5.2 Disease Pressure Index

Entomosporium leaf and berry spot disease pressure is assessed on a real-time basis using a disease pressure index (DPI) equation (Fig. 3.9). Ultimately, DPI values are summed to produce a cumulative DPI value that is used as the basis for control measures. Fry (1982a) described a similar equation that considers the 3 components of the disease triangle for estimating disease severity; however, the DPI equation used in this study is comprised of 4 modules: 1) an assessment of inoculum potential and production, 2) a signal for inoculum release, 3) a calculation of disease development based on the interaction between leaf wetness duration and temperature, and 4) an assessment of host susceptibility. Equation [19] illustrates how the DPI is calculated after each inoculum release event throughout the growing season:

$$Y = A * [B * C] * D \quad [19]$$

where Y is the disease pressure index value, A is an estimation of the amount of inoculum, B signifies inoculum release, C is a calculation for disease development based on leaf wetness duration and temperature after inoculum release, and *D* is an assessment of host susceptibility.

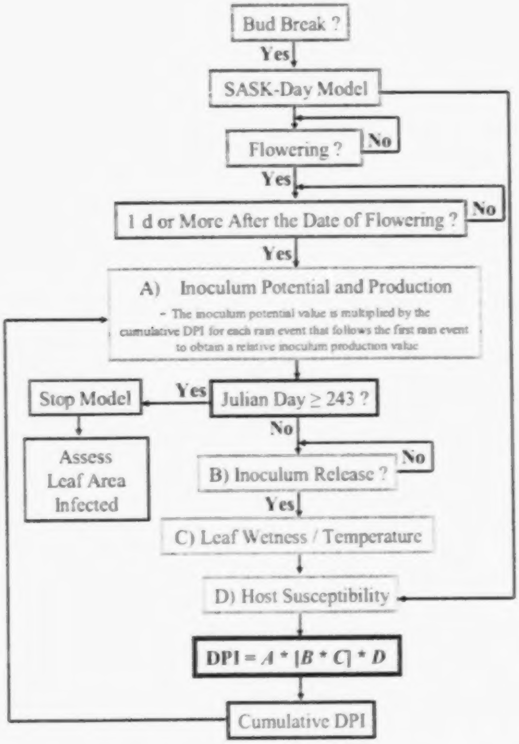
Bud Break ?
Yes
SASK-Day Model
Flowering ?
No
Yes
1 d or More After the Date of Flowering ?
No
Yes
A) Inoculum Potential and Production
- The inoculum potential value is multiplied by the cumulative DPI for each rain event that follows the first rain event to obtain a relative inoculum production value
Stop Model
Yes
Julian Day ≥ 243 ?
No
Assess Leaf Area Infected
B) Inoculum Release ?
No
Yes
C) Leaf Wetness / Temperature
D) Host Susceptibility
DPI = A * [B * C] * D
Cumulative DPI

Figure 3.9 A schematic flow chart illustrating the dynamic disease-forecasting model developed for estimating entomosporium leaf and berry spot disease pressure on saskatoon. The date of bud break (outlined in green), which is entered into the model by the user, starts the phenological component of the disease-forecast model (SASK-Day model). The SASK-Day heat units (outlined in green) are calculated from the date of bud break and used with historical heat unit data to predict the date of flowering (outlined in green) and to estimate axillary vegetative shoot expansion, which in turn is used to estimate host susceptibility (D, outlined in orange). Inoculum potential and production (A, outlined in orange), leaf wetness duration and temperature (C, outlined in orange), and host susceptibility (D, outlined in orange) are used to calculate the disease pressure index (DPI) (outlined in red) after each inoculum release event (B, outlined in orange), which occurs 1 d or more after the date of flowering. After the first rain event that occurs 1 d or more after the date of flowering, a feedback loop mechanism permits the multiplication of the inoculum potential value by the cumulative DPI (outlined in black) for subsequent rain events (see text for explanation). DPI values are summed after each precipitation event to produce a cumulative DPI value. When Julian day 243 (August 31st) has been reached, the model stops calculating DPI and prompts the

user to make an assessment of leaf area infected to obtain an estimate of inoculum potential for the following season.

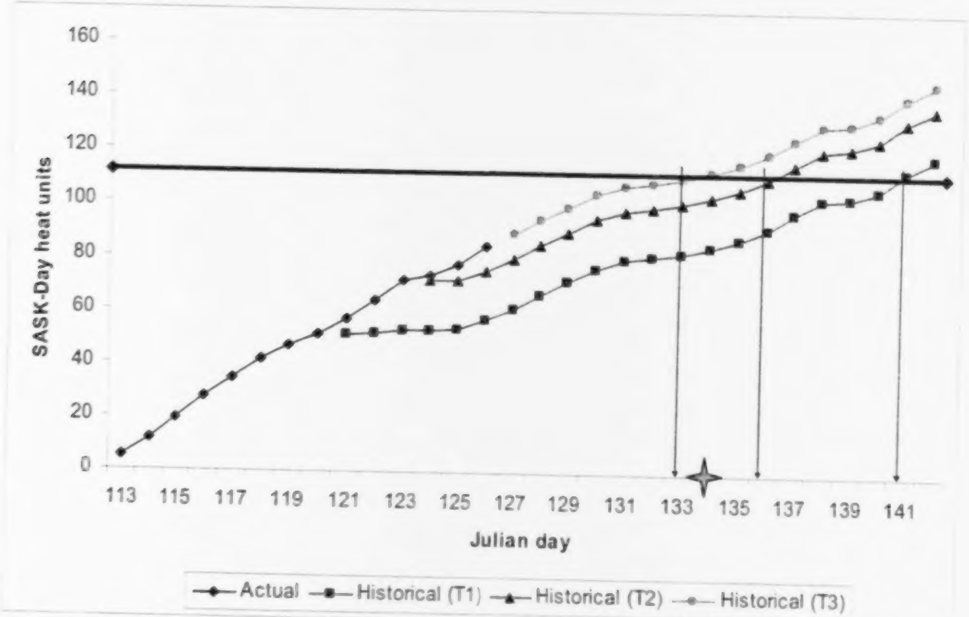

Figure 3.10 Saskatoon physiological day (SASK-Day) and historical daily heat unit data used to predict the date of flowering in a hypothetical simulation. SASK-Day heat units were summed from the time of bud break to produce an estimate of actual heat unit accumulation. From 3 different dates along the curve of actual heat unit data, historical daily heat unit data were used to predict the SASK-Day heat unit sum of 112.1 (indicated by the red horizontal line) which corresponds to the average number of heat units required to reach flowering for the years 1999 to 2001 at the Winnipeg and Carman orchards (see Chapter 3). The actual date of flowering is indicated by the orange star on the x-axis.

3.5.3. Inoculum Potential and Production

Models that do not estimate the amount of overwintering inoculum or consider inoculum production during the growing season are limited for use as an accurate pest management tool. It would be reasonable to expect a lack of disease development when no inoculum is present or introduced into a cropping system. For example, less lesion area develops on *Cucumis sativus* L. when there are lower numbers of *Colletotrichum lagenarium* (Pass.) conidia available to cause infection (Thompson and Jenkins 1985). Given that there is a direct relationship between leaf area that is infected with ELBS disease and inoculum production, then an estimate of orchard inoculum potential can be made providing there is a standardized protocol for disease assessment at the end of the previous growing season (see Appendix C). The use of a standardized protocol for disease assessment has the potential to ensure that an unbiased estimation of the average leaf area infected is calculated (Jones and Windels 1991).

Inoculum potential and production is the first module in the DPI equation (Fig. 3.9, module A). Regression equation [16], originally developed in section 3.4, is used to estimate *E. mespili* inoculum production relative to average percent leaf area infected (PLAI) in an orchard. The inoculum production value was multiplied by a constant of 0.1. This reduced the value calculated for DPI and helped to make the cumulative DPI more suitable for describing observed lesion development when the model was tested in a field trail. This modified regression equation was used to represent a measure of inoculum potential and was used to calculate the DPI for the first rainfall event that occurred after the start of the susceptible period. However, in order to account for the possibility of inoculum build-up after the first rainfall event and a decrease after each fungicide application, a feedback loop was incorporated into the model to estimate fluctuations in inoculum production. This was accomplished by multiplying the inoculum potential value by the cumulative DPI value for all subsequent inoculum release events.

The timing and environmental conditions responsible for *E. mespili* inoculum release in a saskatoon orchard were previously determined. Because conidia (primarily responsible for proliferation of *E. mespili* on saskatoon) are only released during rainfall events, the start of a precipitation event was considered the signal for inoculum release. The value for inoculum release is changed from 0 (no release) to 1 (release) at the start of each precipitation event and therefore permits the calculation of DPI to result in a positive value (Fig. 3.9, module B). When the leaf wetness value that follows the precipitation event falls to 0 then the inoculum release value in the model also returns to 0. The DPI calculated after each rain event is then summed to produce a cumulative DPI value.

3.5.4. Leaf Wetness Duration and Temperature Required for Disease Development

Regression equation [15], which was developed in section 3.3, was used to estimate ELBS disease development on saskatoon leaves based on an 18°C leaf wetness duration and subsequent temperature treatment. Although this model assumes that the environmental conditions required for *E. mespili* conidia germination are the same as those required for disease development, it does provide a basis for estimating disease in relation to leaf wetness duration and temperature. This model used leaf wetness duration and temperature data from the field in order to calculate a value for use in the DPI equation (Fig. 3.9, module C). Average temperature, which was used as the temperature variable (*T*) in the equation, was calculated from temperature data which was collected every 15 min from the start of the inoculum release event until the leaf wetness value returned to 0 after the rainfall event. The value for leaf wetness (*W*), which was used in the equation, is a measure of hours for the same time period used to calculate average temperature. The equation was multiplied by a constant of 0.25 to reduce its value. This helped to reduce the value of the DPI and made the cumulative DPI value more closely reflect observed lesion development in a field test of the model.

3.5.5. Host Susceptibility

Young saskatoon leaves are more susceptible to *E. mespili* infection than older leaves (Ronald et al. 2001). To incorporate this phenomenon into the DPI equation, it was assumed that there is a negative linear relationship between susceptibility and physiological age; however, a future study should be conducted to confirm this assumption. Equation [20] was created to estimate host susceptibility based on physiological development as calculated in Chapter 3:

[20] $$Y = -9.1\text{x}10^{-3}\,X + 5$$

where Y is an estimate of host susceptibility and X represents a measure of physiological development as calculated by the sum of SASK-Day heat units from the date of bud break. The equation was calculated using 2 data points on the assumption that a value of 5 indicates the plant is highly susceptible to *E. mespili* infection at bud break, whereas 1 would indicate that the plant is close to the cessation of axillary vegetative shoot expansion and therefore less susceptible to infection. Shoot expansion cessation occurs when the sum of SASK-Day heat units reaches 441.4. When shoot expansion ceases, the value for host susceptibility will remain at a constant value of 1. The value for host susceptibility is used as the final module in the DPI equation (Fig. 3.9, module D).

3.5.6. Termination of the Calculation of Disease Pressure Index

The dynamic disease-forecasting model will continue to accumulate DPI values on a real-time basis after each precipitation event; however, once Julian day 243 is reached the model is stopped (Fig. 3.9). At this time the model prompts the user to make an assessment of average leaf area infected in the orchard. This value will be used to estimate inoculum potential, which will be used to start the calculation of DPI in the next growing season.

3.5.7. Measuring the Accuracy of the Disease Pressure Index Equation

The accuracy of the DPI equation was evaluated between the date of bud break and fruit harvest. Julian day 190 was selected to represent the start of fruit harvest, as this was the median date for the range of fruit harvest periods observed between 1999 and 2002 at the two orchards used in this study. Unfortunately pertinent weather data were not collected properly at Winnipeg during the 2000 and 2002 growing seasons and at Carman during the 2000 growing season. Therefore, measuring the accuracy of the DPI equation was limited to the Winnipeg 1999 and 2001, and Carman 1999, 2001, and 2002 sites. The DPI values for each site and year, combined years at each site, and the combined sites and years were compared with the observed mean number of lesions per leaf (LESNO) for each week during the pre-harvest period using Pearson's correlation and the coefficient of determination analysis. Before it was possible to make these comparisons, it was necessary to compensate for the lag period between the increase of DPI after each precipitation event and the development of lesions. This period, more commonly known as an incubation period, was 5 d as determined from field observations. Each LESNO measurement was moved back in time 5 d and matched with the cumulative DPI value for that date. Ultimately, combined site and year data were used to create a regression equation that estimates the average number of lesions per leaf given a cumulative DPI value. A visual comparison of the predicted DPI curve relative to the observed LESNO curve over time was also made for each location and year.

The DPI equation produced cumulative values that were highly correlated with observed LESNO for each site and year, combined years for each site, and combined sites and years (Table 3.7). Generally, the DPI equation predicted disease development most accurately at the Winnipeg site. The variation in R^2 values between years was lower at the Winnipeg site (±7% (± standard deviation)) compared to the Carman site (±14%). This result may be explained by the difference in plant health at each site. It was observed that the saskatoon plants in the Carman orchard were notably more stressed than the ones at the Winnipeg orchard. Plant stress in the Carman orchard was induced by a combination of poor drainage, limited nutrients, and a *Cytospora* epidemic, which were all not considered in the disease-forecasting model. The relatively low R^2 value at the Carman 2002 site may have been a result of the short pre-harvest period incurred during this season, which was a result of a very late spring. The late spring in this year seemed to accelerate disease severity early in the growing season, and it did not permit many disease measurements prior to harvest.

When all the data from the sites and years were combined, 82% of the variability for observed disease development could be explained using the DPI equation. Once it was determined that the DPI equation was reliable for predicting disease development, it was possible to create a regression equation that can be used to predict average LESNO in a saskatoon orchard. Linear analysis was performed on the combined data producing an R^2 value of 0.82 (Fig. 3.11). Equation [21] shows the equation derived from the linear regression analysis:

[21] $$Y = 8.0\text{x}10^{-3}X + 1.01$$

where Y represents the predicted average number of lesions per leaf and X is the cumulative DPI value. Using the cumulative DPI together with equation [21] can provide an estimation of LESNO five days in advance of actual symptom development.

The visual comparison of predicted DPI and observed LESNO curves over time showed that the model was generally effective for predicting disease increase prior to fruit harvest at both the Winnipeg and Carman orchards between the years 1999 and 2002

(Figs. 3.12 and 3.13, respectively). The DPI curve more accurately predicted LESNO increase at Winnipeg than in Carman for the years it was tested. This may have been a result of plant stress at the Carman site as indicated above. These figures show that DPI, as estimated by the model, accurately reflects LESNO increase over time in different years and locations.

Table 3.7 Correlation coefficients (r) and coefficients of determination (R^2) between disease pressure index values and the observed number of lesions per leaf during the pre-harvest period. All coefficients were significant at $P \leq 0.01$.

Site	Year(s)	r	R^2
Winnipeg	1999	0.98	0.97
Winnipeg	2001	0.93	0.87
Winnipeg	1999 and 2001	0.96	0.93
Carman	1999	0.96	0.91
Carman	2001	0.86	0.74
Carman	2002	0.80	0.64
Carman	1999, 2001, and 2002	0.91	0.84
All sites and years combined		0.90	0.82

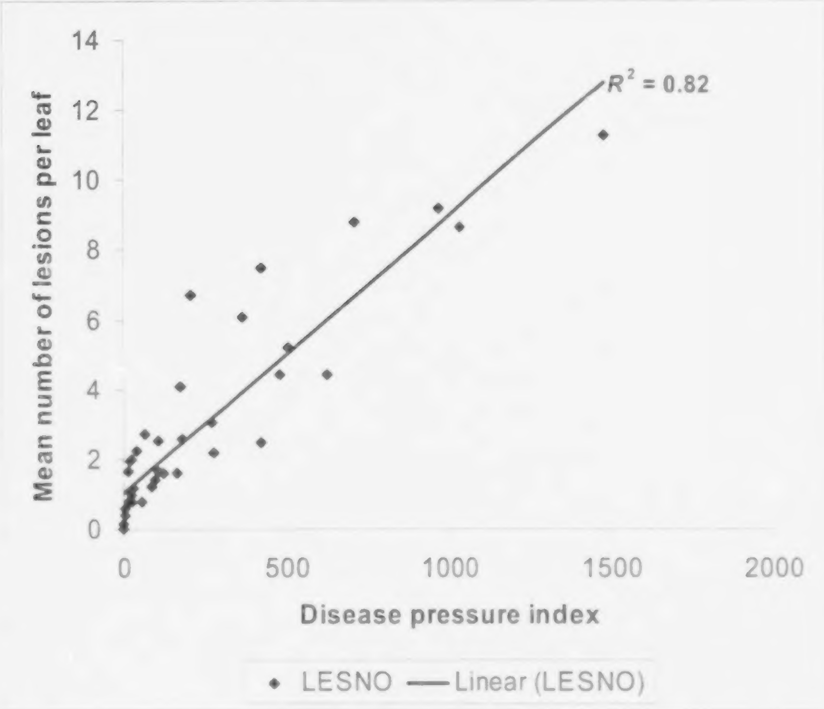

Figure 3.11 Regression analysis between the observed mean number of lesions per saskatoon leaf (LESNO) and predicted disease severity using the disease pressure

index (DPI) equation. Pooled data were collected from the Winnipeg 1999 and 2001, and Carman 1999, 2001 and 2002 sites. Linear regression analysis was performed producing an R^2 value of 0.82.

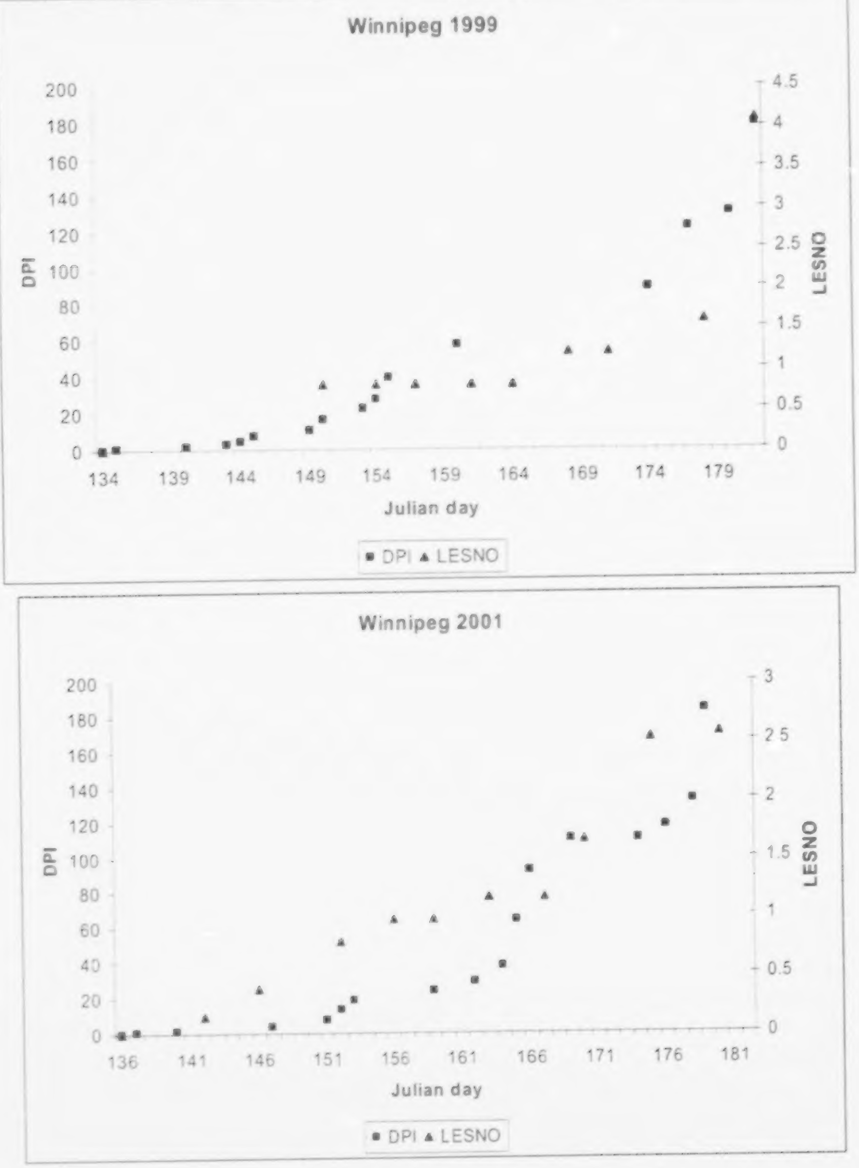

Figure 3.12 Predicted disease pressure index (DPI) increase relative to the observed mean lesion number per leaf (LESNO) over time at the Winnipeg saskatoon orchard during the years 1999 and 2001.

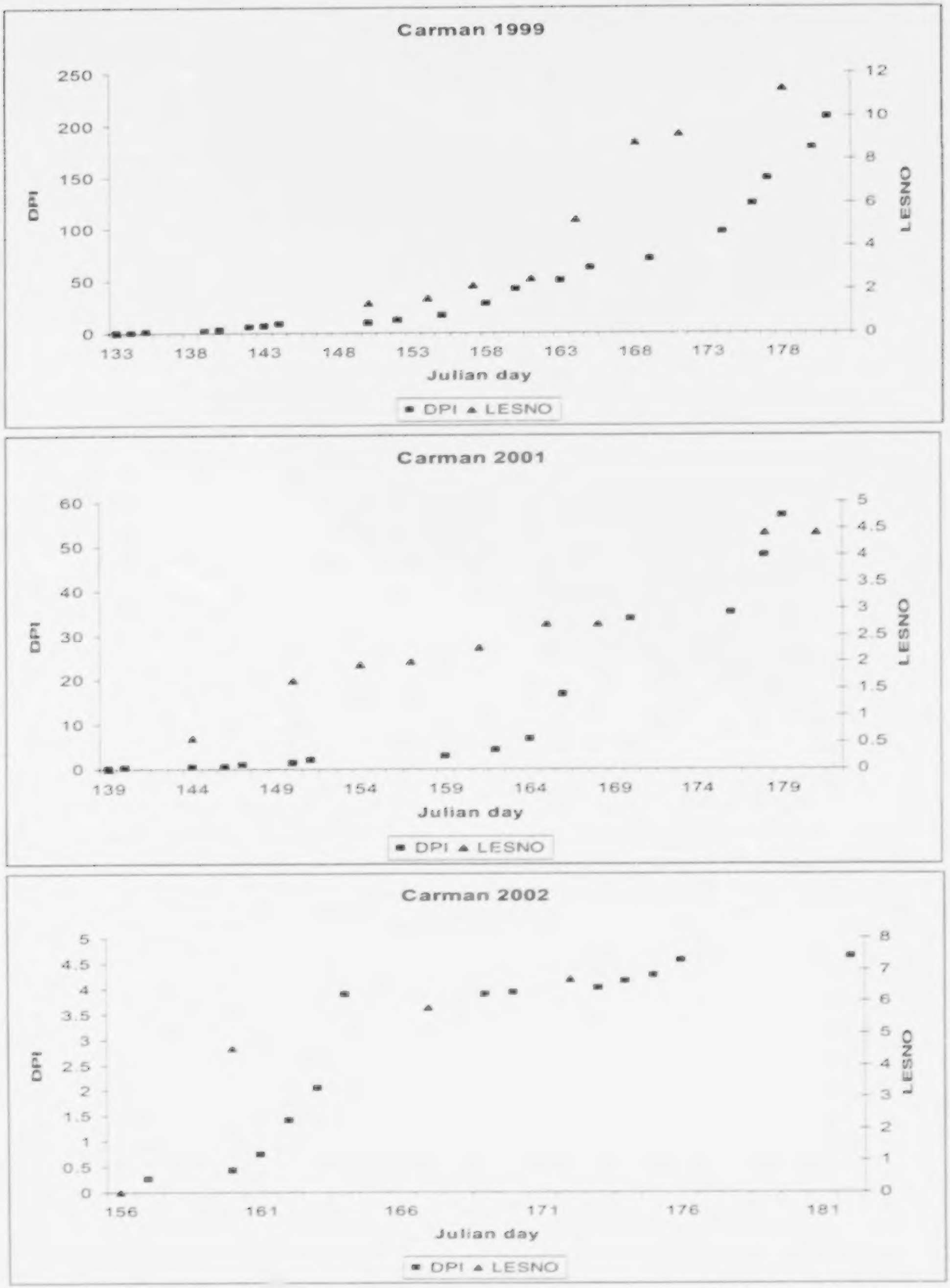

Figure 3.13 Predicted disease pressure index (DPI) increase relative to the observed mean lesion number per leaf (LESNO) over time at the Carman saskatoon orchard during the years 1999, 2001, and 2002.

3.5.8. Incorporating Control Mechanisms into the Disease-Forecasting Model

Control thresholds together with application and control guidelines for Topas 250E were incorporated into the dynamic disease-forecasting model (Fig. 3.9) to create a more comprehensive model that includes disease control capabilities (Fig. 3.14). In the expanded model, once the cumulative DPI produces a positive integer, an assessment can be made to ascertain if a pre-determined control threshold for Topas 250E has been exceeded. For this study, the term control threshold is defined as the level of disease pressure at which a fungicide application may be recommended in order to control the pathogen. Once the first control threshold is exceeded then it is necessary to determine if a spray can be made according to the application guidelines for Topas 250E.

The spray guidelines for Topas 250E indicate that no spray is allowed within 38 d of harvest. Therefore, if the phenological modeling system described above estimates that there are 38 d or less until the date of fruit harvest, then no spray recommendation will be made, despite the possibility of significantly high disease pressure (Fig. 3.14). A future study should be conducted to determine if a contact fungicide like Kumulus can be used to control disease pressure build up when there is less than 38 d to harvest. This may be an important component of the model in the future because it is possible that fruit infection occurs independently of leaf infection. If there are more than 38 d until the predicted date of fruit harvest, or if harvest has already occurred (entered by the grower into the model), then the model will consider if Topas 250E was applied previously. If there were no previous fungicide applications then the model will suggest an application of Topas 250E; however, if Topas 250E was previously applied, then the model determines if the fungicide application is still active. While the axillary vegetative shoots are still expanding (SASK-Day <441.4), the model will not suggest additional spray applications for a period of 14 d after a previous spray application unless it is the 39th day before the predicted date of fruit harvest. The 39th day before fruit harvest is the last possible day that Topas 250E can be applied. If it is the 39th day before fruit harvest and the second control threshold is exceeded, the model will recommend another application of Topas 250E despite the fact that fewer than 14 d may have elapsed since the last spray. If the control threshold is not exceeded on the 39th day before fruit harvest the model will not recommend another spray application; however, a future study should be designed to examine the benefit of a protective Topas 250E spray application on the 39th day before fruit harvest. Once the axillary vegetative shoots cease to expand (SASK-Day >441.4) it is assumed (based on the spray guidelines for Topas 250E) that the spray applications are effective for controlling *E. mespili* for a period of 21 d. If the model determines that the last application of Topas 250E is not active then it will consider if the second control threshold has been exceeded. If it has been exceeded, the model will recommend another spray of Topas 250E.

Each time Topas 250E is applied, the cumulative DPI value in the model is reset to 0.1 (Fig. 3.14). Because the cumulative DPI is reset to 0.1, the inoculum potential value is reduced as a result of a feedback loop. After each spray application, the number of days is counted in order to keep track of the fungicide active period. DPI values will continue to be summed from the date of Topas 250E application. If no fungicide is applied, then the model will look for next inoculum release event and subsequently DPI will continue to accumulate.

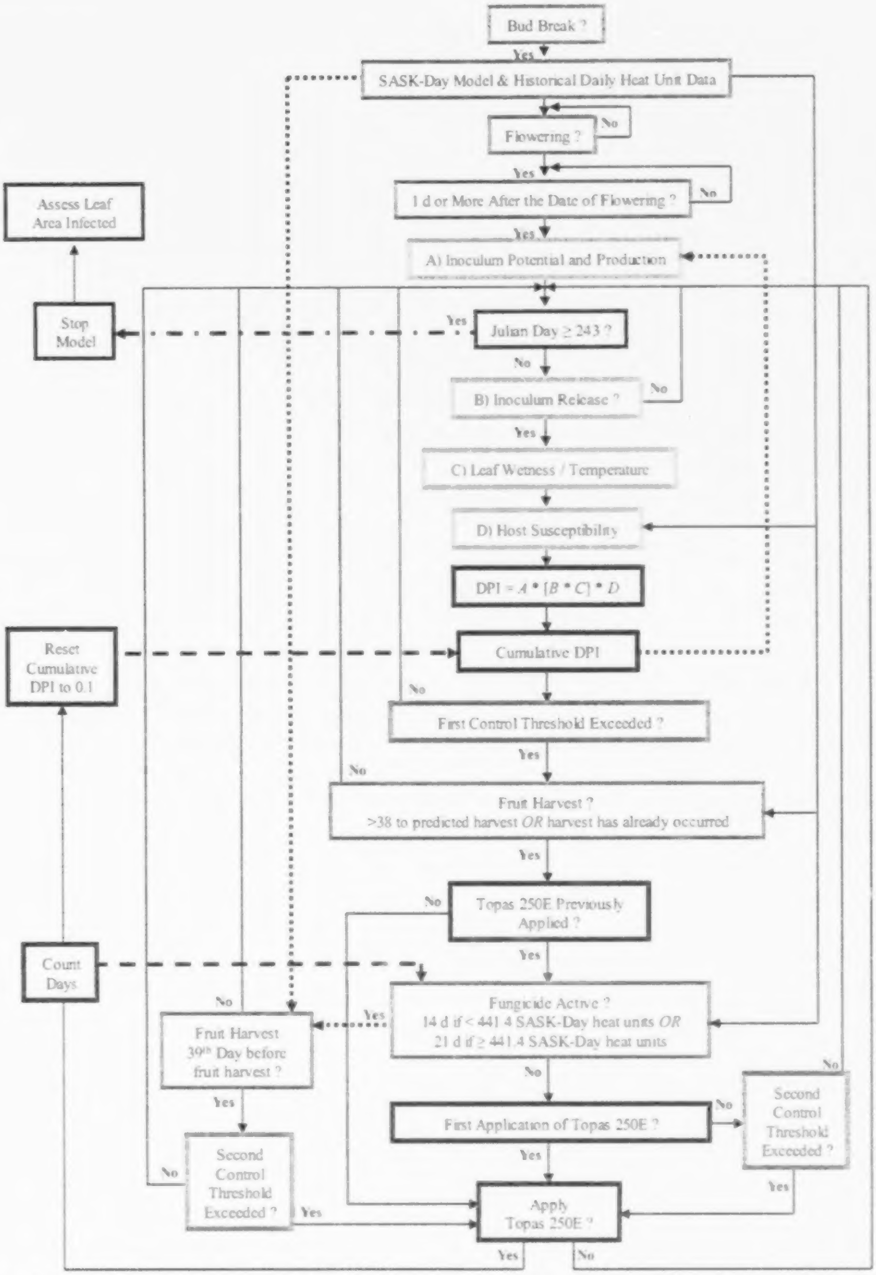
Bud Break ?
Yes
SASK-Day Model & Historical Daily Heat Unit Data
Flowering ?
No
Yes
1 d or More After the Date of Flowering ?
No
Yes
Assess Leaf Area Infected
A) Inoculum Potential and Production
Stop Model
Yes
Julian Day ≥ 243 ?
No
B) Inoculum Release ?
No
Yes
C) Leaf Wetness / Temperature
D) Host Susceptibility
DPI = A * [B * C] * D
Reset Cumulative DPI to 0.1
Cumulative DPI
No
First Control Threshold Exceeded ?
Yes
No
Fruit Harvest ?
>38 to predicted harvest OR harvest has already occurred
Yes
No
Topas 250E Previously Applied ?
Yes
Count Days
No
Fungicide Active ?
14 d if < 441.4 SASK-Day heat units OR
21 d if ≥ 441.4 SASK-Day heat units
Yes
Fruit Harvest 39th Day before fruit harvest ?
No
Yes
No
First Application of Topas 250E ?
No
Second Control Threshold Exceeded ?
Yes
No
Second Control Threshold Exceeded ?
Yes
Yes
Apply Topas 250E ?
Yes
No

Figure 3.14. A dynamic disease-forecasting model for entomosporium leaf and berry spot on saskatoon that incorporates control thresholds and Topas 250E application and control guidelines.

3.5.9. Field Testing the Model

During the summer of 2002 a field test was conducted at the Carman orchard to evaluate the effectiveness of the dynamic disease-forecasting model. The orchard was divided into 5 sections: 3 sections received no fungicide applications and 2 received fungicide treatments as specified by the disease-forecasting model. Because disease history in the orchard was severe with an average percent leaf area infected (PLAI) value of 42, the modified inoculum production regression equation calculated the inoculum potential value to the maximum value of 0.22. The control thresholds were conservatively set in the first test of the disease-forecasting model. Zero was used for the first control threshold and 0.1 was used as the second control threshold. Fungicide applications were applied with a backpack sprayer following the manufacturer's application guidelines (Engage Agro Corporation, Guelph, Ontario). An assessment of disease severity was made weekly between Julian day 158 and 237 by measuring the average PLAI of 5 leaf samples from each of 5 randomly selected plants in each orchard section.

The SASK-Day model together with historical heat unit data were used to predict the dates of flowering and fruit harvest from the date of bud break, which occurred on Julian day 116. This system successfully predicted flowering as early as 25 d after the date of bud break, which was 9 d in advance of the actual flowering date (Julian day 150), and fruit harvest 25 d after bud break, which was 57 d in advance of the actual fruit harvest date (Julian day 204). The first positive DPI value (0.26) was calculated on Julian day 157, which was 6 d after the start of the susceptible period. As a result, Topas 250E was applied for the first time on Julian day 158. The spray was permitted because there were 46 d until the predicted fruit harvest date, which occurred on Julian day 204. A second Topas 250E application was applied 7 d later on Julian day 165, because the cumulative DPI had risen to 3.73, exceeding the 0.1 control threshold for the 39th day before the predicted date of fruit harvest.

The average PLAI in the treated sections of the orchard was limited to 0.48% by the start of fruit harvest and was kept below 0.6% until fruit harvest was complete (Fig. 3.15). PLAI in the untreated sections of the orchard averaged 1.31% by the start of fruit harvest and reached 4.98% by the end of fruit harvest. According to paired t-test analysis, average PLAI was significantly higher by the time fruit harvest began ($P \leq 0.05$). Moreover, additional benefits in disease reduction were realized after fruit harvest when disease levels began to increase exponentially in the untreated sections of the orchard, resulting in an average PLAI of over 50% by Julian day 237. The average PLAI in the treated sections of the orchard remained below 1% throughout the entire sampling period of this study. It is hypothesized that ELBS disease pressure should be suppressed in the following growing season due to reduced inoculum production. This is primarily because less infected leaf litter will overwinter. The lower disease pressure should also limit the number of fruit and young shoot infections, which can also be a source of inoculum in the next season.

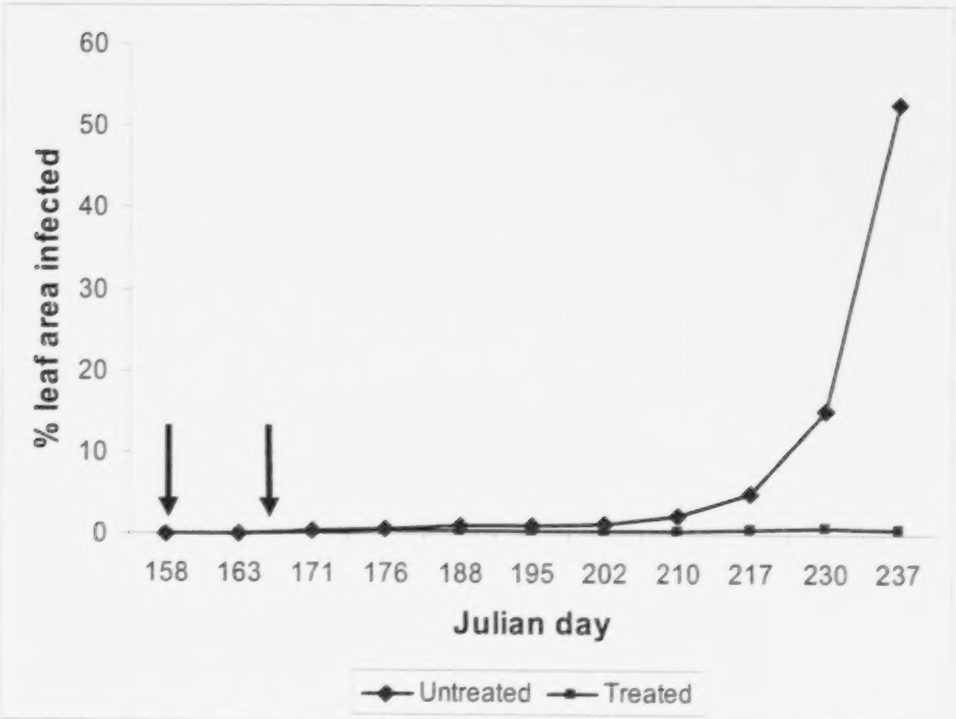

Figure 3.15 Increase of average percent leaf area infected over time for fungicide treated and untreated sections at the Carman orchard in 2002. The treated section of the orchard received 2 Topas 250E applications (arrows) based on recommendations from the dynamic disease-forecasting model. Percent leaf area infected after Julian day 204 (start of fruit harvest) was significantly greater in the untreated section of the orchard and exceeded 50% by the end of the season. Conversely, in the treated section, percent leaf area infected did not exceed 1% at any point between Julian day 158 and 237.

3.5.10 Discussion

Comparing the R^2 values calculated in this study against other disease modeling studies can provide an assessment of model accuracy. In a controlled environment study, Grove (2002) described the majority of variation in disease severity on cherry ($R^2 = 0.80$) and peach ($R^2 = 0.83$) foliage using models based on leaf wetness duration and temperature. In another model, Asher and Williams (1991) were able to account for 95% of the variation in powdery mildew on sugar beet based on regression analysis with temperature and precipitation data in the period April to August. A degree-hour model was used to describe 80% of the observed infection variability of *Puccinia graminis* subsp. *graminicola* Z. Urb. on perennial ryegrass seed crops (Pfender 2003). The authors of all three studies concluded that the coefficient of determination values for each model were acceptable for describing disease development. Therefore, it seems appropriate to conclude that the dynamic-disease forecasting model developed in this study is sufficient for predicting ELBS disease development on saskatoon.

Entomosporium leaf and berry spot disease at the Carman 2002 site was effectively controlled using the dynamic disease-forecasting model. The model was able to successfully predict disease symptom development 5 d in advance of actual occurrence. Only 2 applications of Topas 250E were suggested by the disease-forecasting model compared to the 3 applications that are currently recommended based on plant development stages. Even though the coefficients of determination between DPI and LESNO accounted for only 64% of the variation in disease levels in the untreated sections of the Carman 2002 site, the model effectively controlled ELBS disease on the saskatoon plants prior to, and after, fruit harvest. Given that 0.64 was the lowest observed coefficient of determination between DPI and LESNO for all the sites and years, it was concluded that spray recommendations based on the model are sufficient for controlling *E. mespili* in 'Smoky' saskatoon orchards located in Manitoba. If in a future study it is shown that disease on saskatoon fruit is not controlled using the current version of the model, then protocol for Kumulus applications could be added to the model.

A reduction in disease development prior to and during fruit harvest should help to ensure a high yielding and high quality fruit crop. Johnson (1988) and Madden et al. (1981) used models to quantitatively describe the effects of disease on yield loss and generally found that as disease severity increased so does the loss in yield. Conway et al. (1990) showed how various fungicides applied before harvest can be used to secure greater yield by reducing *Cercospora asparagi* Sacc. proliferation on asparagus. However, in order to verify this concept in saskatoon orchards a future study is necessary to assess the relationship between infected leaf and fruit area. A prerequisite for such a study is to determine the timing of rain splashed inoculum dispersal. For example, despite prolific early season leaf infection, little disease may develop on saskatoon fruit if no inoculum is released during the fruit maturation period. Moreover, the study should identify if fruit infection can occur independent of leaf infection, and whether young or mature fruit are more susceptible to infection.

Results from the test of the dynamic disease-forecasting model indicated that if symptom development can be properly controlled early in the season then disease levels may remain minimal throughout the growing season. This response appears to be linked to the availability of early season inoculum. The amount of early season inoculum is a major limiting factor affecting powdery mildew epidemics (Asher and Williams 1991). Similarly, the success of apple scab disease is reliant on early season inoculum and infection because that is when the tree is most susceptible (Boone 1971). In potatoes, given that late blight epidemic development is proportional to the rate of increased inoculum production and dispersal as well as the viability and infectivity of the inoculum (Bashi et al. 1982), early season disease control is fundamental in limiting the proliferation of *Phytophthora infestans* (Mont.) de Bary. Less area under a disease progress curve suggests that there is minor plant stress, which in turn can be correlated with higher yield levels (Madden et al 1981). Reductions in disease levels after fruit harvest should help to improve saskatoon plant vigour and in doing so will have the potential to increase harvestable yields and limit ELBS disease development in subsequent years. A future study should be conducted to examine the long-term effects of effective disease control on saskatoon vigour and harvestable yield, as well as, viable *E. mespili* inoculum production.

In conclusion, a DPI equation was created and used to predict the development of ELBS disease on 'Smoky' saskatoon. The DPI equation integrated information regarding saskatoon phenological development and the relationship of disease to inoculum potential and production, leaf wetness duration and temperature, inoculum release, and host susceptibility. Cumulative DPI values were used to successfully predict disease severity increase 5 d prior to symptom development in two orchards over several years. Because the model was successful in predicting disease symptom development, control thresholds together with Topas 250E application and control guidelines were added to the model and shown to be effective for controlling disease in a field trial. The model used zero disease tolerance control thresholds to recommend 2 fungicide spray applications, which is one less spray than what is currently recommended. Although zero tolerance disease control thresholds were used in this study, economic thresholds should be established for each geographic region and cultivar to help identify the point where the cost of control equals the value of crop loss if no action is taken. To date, no study has investigated the relationship between disease severity on leaves and fruit of saskatoon. A properly set economic threshold may help to further reduce the number of Topas 250E applications and still ensure optimal fruit yield. Currently, the disease-forecasting model incorporates only temperature, leaf wetness, and precipitation as weather parameters in calculating DPI. In future versions of the model it may be possible to include additional weather parameters that influence disease development such as relative humidity, solar radiation (Baudoin 1986a), and wind speed to improve model accuracy.

3.6. General Discussion And Conclusions

The temperature parameters of the potato physiological day model were modified to create the saskatoon physiological day (SASK-Day) model used to predict the phenological events of flowering, fruit harvest, and the cessation of axillary vegetative shoot expansion from the date of bud break. Based on a comparison of standard deviation values represented in days, the SASK-Day model proved to be more precise than the growing degree-day and Julian day systems for predicting phenological development of saskatoon. When combined with historical heat unit data, it was possible to predict flowering and fruit harvest well in advance of actual occurrence. The phenological stage of flowering can be used as a reference point for the application of pesticides like Decis 5.0EC to control insect pests like apple curculio, hawthorn weevil, lygus bug, saskatoon bud moth, saskatoon sawfly, and tarnished plant bug. A phenological model that predicts the date of fruit harvest provides growers with the time to plan harvest operations in advance so that fruit harvest can be optimized. The heat units required for the cessation of axillary vegetative shoot growth were used to create a negative linear model to estimate how the susceptibility of saskatoon leaves decrease relative to increasing physiological time. Future work is required to verify that host susceptibility decreases in a linear fashion relative to the accumulation of SASK-Day heat units.

An enhanced understanding of the epidemiology of *Entomosporium mespili* (D.C.) Sacc. and its relationship with *Amelanchier alnifolia* Nutt. (saskatoon) was gained from this research. Leaf wetness and temperature were identified as two important weather parameters that interact to affect the infection success of conidia on saskatoon leaves, and regression analysis was used to characterize this relationship. It was confirmed that young saskatoon leaves are significantly more susceptible to infection than older, more physiologically developed leaves when subjected to specific combinations of leaf wetness duration and temperature. Once regression analysis illustrated that there was a positive linear relationship between percent leaf area infected and inoculum production, a disease assessment protocol was established to estimate inoculum potential for the next growing season. Using a splash dispersed conidia trap, it was confirmed that conidia of *E. mespili* were not released into the air, but rather into rainwater. The start of the susceptible period corresponded with the first precipitation event that occurred 1 d or more after the date of flowering. Based on the time it took for the first disease symptoms to appear, the incubation period for early season disease development was 5 d. Although no conidia were captured until the start of exponential infected leaf area increase, it is still believed that they are responsible for causing initial disease symptom development. The role of ascospores in causing initial and subsequent disease symptom development was not identified in this study but should be investigated in the future. It is possible that ascospores play a role in causing initial entomosporium leaf and berry spot (ELBS) disease development in geographically isolated orchards. In precipitation events that caused inoculum release, conidia were primarily captured in the first 2 h of rainfall. Multiple late season rain events can promote disease level increase by stimulating conidia release.

The SASK-Day phenological model and epidemiological data were combined to create a dynamic disease-forecasting model for ELBS disease on saskatoon. A disease pressure index (DPI) equation was formulated and its output compared to observed disease development in two different orchards over several years. The equation proved to be reliable for estimating disease level increase for the period between bud break and fruit harvest. The DPI was not considered beyond this point because disease development after fruit harvest does not affect crop yield in the current growing season. Temperature, leaf wetness, and precipitation data were sufficient for describing most of the variation in observed disease levels and thus were considered the primary weather parameters influencing disease development.

In an effort to increase the functionality of the disease model, control thresholds, along with application and control guidelines for the fungicide Topas 250E, were incorporated into the model. A step-by-step look at the dynamic disease-forecasting model is provided in Appendix D. Disease was controlled when conservative control thresholds were used in a field test of the dynamic disease-forecasting model. Compared to current spray recommendations, the model was able to achieve good disease control using one less Topas 250E application. Conservative control thresholds and a Topas 250E application after the first precipitation event at the start of the susceptible period were the most important factors influencing disease control. Although no association was made between disease severity on leaves and fruit, it may be reasonable to expect little fruit infection if leaf infection is minimized early in the season. A future study should be conducted to examine the relationship between infected leaf area and fruit infection. It was also observed that if ELBS were properly controlled early in the season then disease severity would be minimal at the end of the growing season. Future work is required to determine the benefit of disease control on fruit yield and plant vigour in subsequent seasons.

The model advanced current fungicide application practices by determining the first and last date that a spray can be used before fruit harvest. Furthermore, it provided the means to determine if a spray is required after harvest to help control disease build up late in the season. It was realized after the construction and field evaluation of the dynamic disease-forecasting model that the model could be simplified. In essence, if a statement can be made regarding the presence of inoculum, then Topas 250E should be applied according to a control threshold between the start of the susceptible period and the 39th day before the predicted date of fruit harvest. The number of fungicide applications could be reduced if it can be determined that higher control thresholds can be tolerated. Therefore, future work is required to establish such economic thresholds for control when validating the model on different cultivars and geographic regions.

Unlike humans, complex biological systems in nature must rely on the relationship between cause and effect rather than choice to react to the environment. Therefore, it is possible to predict the outcome of such biological systems mathematically, provided there is an understanding of the interacting elements. Ultimately, this provides the mean to forecast and control these biological phenomena.

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3.8. Appendices

Appendix A: Schematics of a Splash Dispersed Conidia Trap used to Collect *Entomosporium mespili* Conidia

A novel conidia trap was constructed to capture splash dispersed *Entomosporium mespili* conidia as they are disseminated in rainwater. The conidia trap was constructed from a clear 40 cm piece of PVC piping with a fixed bottom and lid, a plastic funnel, a stainless steel carousel machined to hold 20 test tubes, twenty 50 ml test tubes, and a step electrical motor (Fig. A.1). A runoff drain was inserted at the base of the splash dispersed sport trap unit to release excess water that may overflow from the test tubes during heavy rainfall events. Power was supplied from a 12 volt deep-cycle battery, which was charged from a 30-watt solar panel.

Before the conidia trap was placed in the field, the funnel was treated with a canola based cooking spray to reduce the surface tension of rainwater on the surface of the funnel. The funnel siphons rainwater as it moves through the plant canopy into a 50 ml test tubes positioned on the turntable beneath the funnel (Fig. A.1). Test tubes were rotated one position on the carousel in 1 h intervals. Two milliliters of glycerol were placed in each test tube. The glycerol helped to reduce water evaporation from the test tube and inhibit conidium desiccation prior to collection.

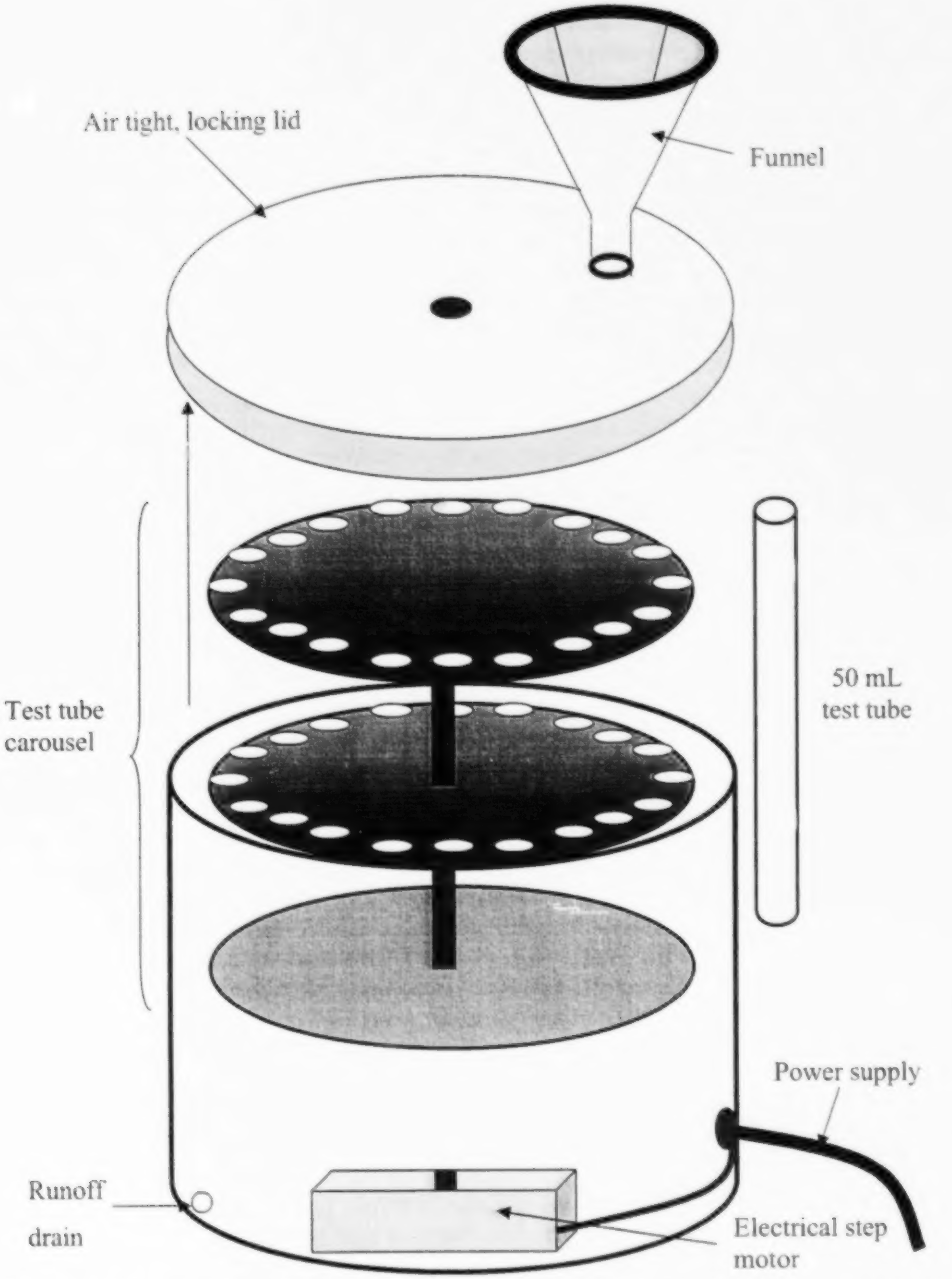
Air tight, locking lid
Funnel
Test tube carousel
50 mL test tube
Power supply
Runoff drain
Electrical step motor

Figure A.1 Schematics of a splash dispersed conidia trap used to capture rainwater disseminated conidia of *Entomosporium mespili* within a saskatoon plant canopy. The conidia trap was designed and built at the University of Manitoba.

Appendix B: Initial Disease Symptom Development in the Field Relative to Rainfall and the Phenological Event of Flowering

Initial disease symptom development, rainfall and flowering data were collected from Winnipeg and Carman saskatoon orchards between the years 1999 and 2002 (Table B.1). Initial disease symptoms appeared 5 d after the first rainfall event that occurred 1 d or more after the date of flowering in almost every site and year. Only in 1999 at Carman were there more than 5 d between initial disease symptom development and the first rainfall event that occurred 1 d or more after the date of flowering. This discrepancy was likely a result of delayed field sampling. Field sampling was delayed due to rainfall and the remote location of the field.

Table B.1 Initial disease symptom development in the field relative to rainfall and the date of flowering at the Winnipeg and Carman orchards between the years 1999 and 2002.

Winnipeg			
1999 Julian Day	2000 Julian Day	2001 Julian Day	2002 Julian Day
█ ▨	█ ▨	█	█
░ 127	░ 133 ▨	░ 134 ▨	░ 151
128	134	135 ■	152
129	135	136 ▨	153
130	136	137	154
131 ■	137	138	155
132	138	139 ▨	156 ■
133 ▨	139	▒ 140	157
134 ▨	140		158 ▨
135 ▨	141 ■		159 ▨
▒ 136	142 ▨		160 ▨
	143		▒ 161
	144		
	145		
	▒ 146		

Key	
Flowering	█
One day after flowering	░
First rainfall event 1 d or more after the date of flowering	■
Rainfall	▨
Initial disease symptom development	▒

Carman			
1999 Julian Day	2000 Julian Day	2001 Julian Day	2002 Julian Day
█	█	█ ▨	█ ▨
░ 133	░ 137	░ 138	░ 151
134 ■	138	139	152
135 ▨	139	140 ■	153
136	140	141	154
137	141	142 ▨	155
138	142	143 ▨	156
139 ▨	143	144 ▨	157 ■
▒ 140	144 ■	▒ 145	158
	145		159
	146		160 ▨
	147		161 ▨
	148		▒ 162
	▒ 149		

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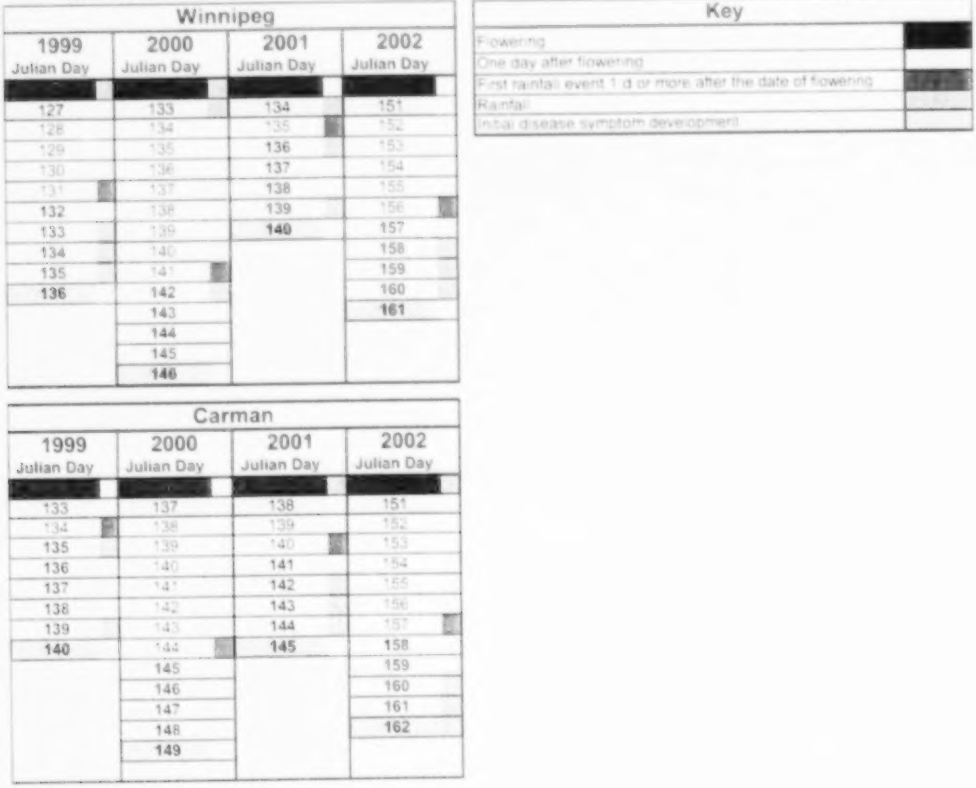


Winnipeg			
1999 Julian Day	2000 Julian Day	2001 Julian Day	2002 Julian Day
■	■	■	■
127	133	134	151
128	134	135 ▨	152
129	135	136	153
130	136	137	154
131 ▨	137	138	155
132	138	139	156 ▨
133	139	**140**	157
134	140		158
135	141 ▨		159
136	142		160
	143		**161**
	144		
	145		
	146		

Carman			
1999 Julian Day	2000 Julian Day	2001 Julian Day	2002 Julian Day
■	■	■	■
133	137	138	151
134 ▨	138	139	152
135	139	140 ▨	153
136	140	141	154
137	141	142	155
138	142	143	156
139	143	144	157 ▨
140	144 ▨	**145**	158
	145		159
	146		160
	147		161
	148		**162**
	149		

Key	
Flowering	■
One day after flowering	□
First rainfall event 1 d or more after the date of flowering	▨
Rainfall	░
Initial disease symptom development	□

Appendix C: Standardized Assessment of Average Percent Leaf Area Infected in an Orchard

The assessment of inoculum production depends on input from the grower. It was determined in Chapter 5 that an important component of the regression equation used to predict inoculum production is an assessment of disease development. Thus, to facilitate disease assessment, a standard protocol for all growers had to be created.

An estimate of inoculum potential is based on an assessment of the average percent leaf area infected (PLAI) at the end of the growing season. The assessment of average PLAI at this time is intended to provide an estimation of how much inoculum is overwintering and will subsequently be available for early season infections during the next season. For the grower to be able to assess the disease in the orchard, there are 3 prerequisites: 1) determine the number of plants to sample; 2) understand sampling procedures; and 3) score the average PLAI.

1) Number of sample plants. As long as the sampling is not biased, as sample size increases, the likelihood of an incorrect assessment of average PLAI will decrease (Jones and Windels 1991). It is suggested that in an orchard containing 1000 plants, a fifty-plant-sample should be sufficient for assessing average PLAI. This sample number will provide 100 individual measurements of disease when two leaf samples are randomly selected 1 m from the ground on two sides of each randomly selected plant. If the orchard is larger than 1000 plants, the sampling number is adjusted accordingly to provide a good representation of average PLAI.

2) Sampling procedures. Sampling should be done on or soon after Julian day 243 (August 31st). When assessing PLAI in an orchard it should be free from moisture to ensure that no inoculum is spread during sampling. A modified, X-shaped field-sampling pattern, used by Jones and Windels (1991) to monitor cercospora leaf spot of sugar beet, is ideal for getting a good representation of disease levels throughout a field (Fig. C.1).

3) Scoring PLAI. Growers must be able to recognize the characteristic symptoms of entomosporium leaf and berry spot disease for field-monitoring activities. Using Assess for Windows (APS Press, Saint Paul, MN), a scoring system was developed to estimate 10 different levels of PLAI severity (Fig. C.2). In Figure C.2, the top row represents leaves with relatively low disease severity and the lower row represents leaves with much greater levels of infection. If a heavily infected leaf sample falls between two damage categories then the higher damage category should be selected. The disease severity rating for each leaf sample is then recorded in tabular form (Table C.1) and used to produce an estimate of average PLAI in the orchard.

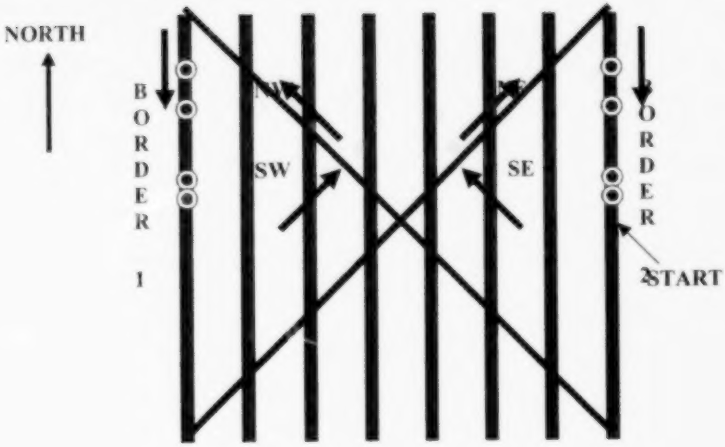
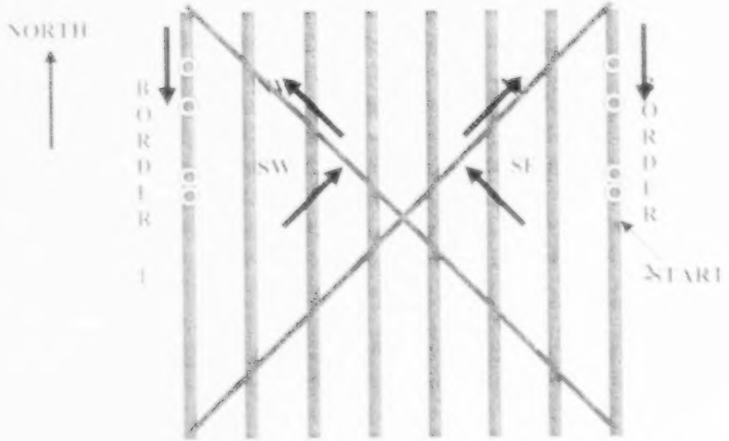


Figure C.1 A proposed field-monitoring plan for determining the average percent of saskatoon leaf area infected. The green lines represent saskatoon plant rows. Where the red lines or target symbols overlap these green lines is where an assessment of disease severity should be made. The field can be divided into 6 sampling areas: Southeast, Northwest, Border 1, Southwest, Northeast, and Border 2. If sampling begins in the Southeast corner of the field and proceeds along the arrows as shown, a complete sampling circuit of the field can be done without having to backtrack over previously sampled areas.



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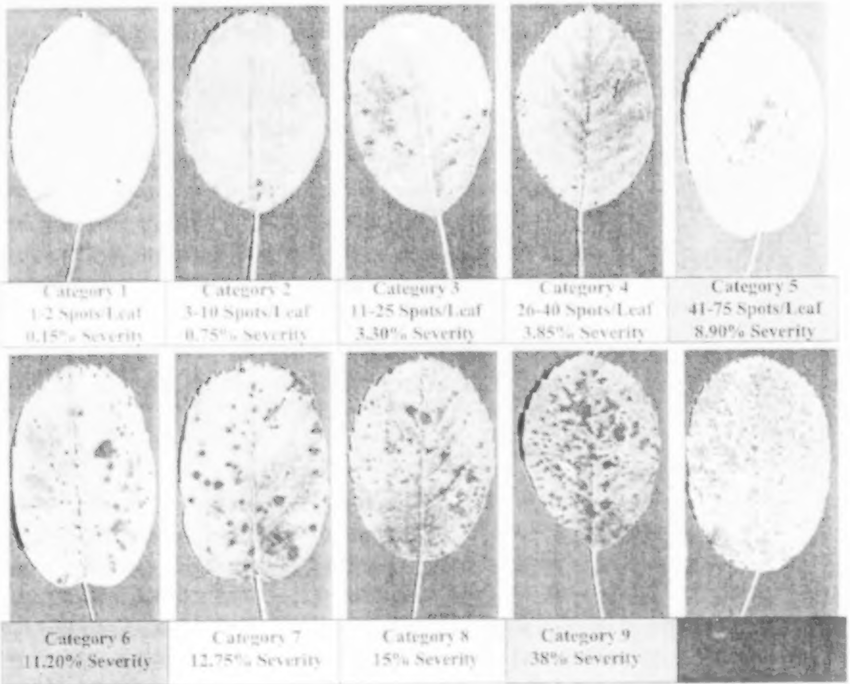
Category 1
1-2 Spots/Leaf
0.15% Severity
Category 2
3-10 Spots/Leaf
0.75% Severity
Category 3
11-25 Spots/Leaf
3.30% Severity
Category 4
26-40 Spots/Leaf
3.85% Severity
Category 5
41-75 Spots/Leaf
8.90% Severity
Category 6
11.20% Severity
Category 7
12.75% Severity
Category 8
15% Severity
Category 9
38% Severity

Figure C.2 A series of photographs of saskatoon leaves with varying degrees of entomosporium leaf and berry spot representing 10 different disease severity categories. This figure is should be used to assess the disease severity of leaf samples.

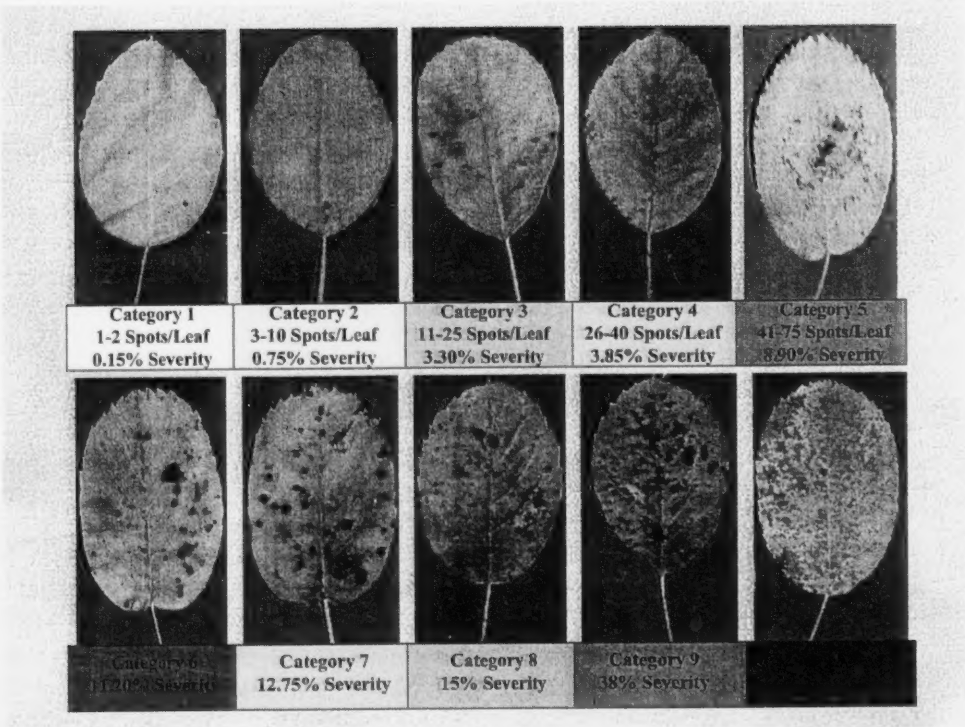
Category 1
1-2 Spots/Leaf
0.15% Severity
Category 2
3-10 Spots/Leaf
0.75% Severity
Category 3
11-25 Spots/Leaf
3.30% Severity
Category 4
26-40 Spots/Leaf
3.85% Severity
Category 5
41-75 Spots/Leaf
8.90% Severity
Category 6
11.20% Severity
Category 7
12.75% Severity
Category 8
15% Severity
Category 9
38% Severity

Figure C.2 A series of photographs of saskatoon leaves with varying degrees of entomosporium leaf and berry spot representing 10 different disease severity categories. This figure is should be used to assess the disease severity of leaf samples.

Table C.1 A disease level assessment table for calculating the average percent of saskatoon leaf area that is showing symptoms of entomosporium leaf and berry spot disease. The damage category of leaves in each location of the orchard is determined using Figures B.1 and B.2. For each leaf, one tally representing the leaf's damage rating is placed under the corresponding position in the field. Tallies in each damage category are summed and then multiplied by the corresponding % severity value in the table to obtain a leaf severity value. To calculate the average percent of infected leaf area (PLAI) for the orchard, the sum of leaf severity is divided by the sum of disease observations (tallies).

	Position in field								
Damage category	SE	NW	Border	NE	SW	Border	Total	% severity	Leaf severity
0								0	
1								0.15	
2								0.75	
3								3.30	
4								3.85	
5								8.90	
6								11.20	
7								12.75	
8								15	
9								38	
10								42	
								<-- Sum -->	

PLAI = Sum of leaf severity / Sum of tallies-->

Appendix D: Sequence of Steps in the Dynamic Disease-Forecasting Model

The dynamic disease-forecasting model (Fig. 6.6) was integrated with the spreadsheet software program Excel (Microsoft, Bellevue, WA) to calculate diseases pressure index (DPI) using hourly temperature, leaf wetness and precipitation data. The sequences of steps in the model can be incorporated into a computer software program using a standard programming language such as Java. The following outlines the procedural sequence of steps in the model.

1) Prior to the start of the growing season the user must enter a percent leaf area infected (PLAI) value into the model. Guidelines for calculating average PLAI in an orchard are found in Appendix C. This measurement will be used to calculate the 'Inoculum Potential' value in the model.
2) The user should enter the date of bud break into the model when more than 50% of the reproductive buds have opened to reveal their silver pubescent hairs (see Fig. 3.1A).
3) From the date of bud break, the saskatoon physiological day (SASK-Day) model (a component of the dynamic disease-forecasting model) uses daily maximum and minimum temperatures to calculate daily heat unit accumulation (see Equations 7-11). Daily SASK-Day heat units are calculated every twenty-four hours at 12 mid-night, starting on the date of bud break.
4) The SASK-Day model is used to calculate the 112.1 heat units required to reach the phenological stage of flowering. The date of flowering can also be entered directly into the model by the user. The phenological stage of flowering is defined as the time that the petals of the apical flower in the inflorescence are completely unfolded to expose the reproductive structures of the flower (see Fig. 3.1B).
5) Twenty-four hours after the date of flowering, the model automatically calculates 'Inoculum Potential' using the average PLAI value, which was entered into the model at the start of the growing season (see Equation [16]).
6) At this point, before the model begins to calculate disease pressure index (DPI) it considers the Julian date. If it is Julian day 243 or greater then the model stops calculating DPI and prompts the user to make an assessment of PLAI in the orchard. If the model determines that it is not yet Julian day 243 then it waits for an 'Inoculum Release' event (rainfall).
7) The value for 'Inoculum Release' remains at 0 until a rainfall event occurs. When rain begins to fall, the 'Inoculum Release' value is changed from 0 to 1 and remains at a value of 1 until the leaf wetness value that occurs with the rainfall event returns to 0.
8) At the end of the leaf wetness period the 'Leaf wetness/Temperature' regression equation (see Equation [15]) is used to calculate a value for disease development using variables for temperature (T) and leaf wetness (W). The variable for T represents the average temperature during the leaf wetness period, and the variable for W represents a measurement of the leaf wetness duration in hours.
9) After a value for disease development relative to the average temperature and leaf wetness duration has been calculated, a value for 'Host Susceptibility' is determined using equation [20]. Equation [20] uses the sum of SASK-Day heat

units, which were calculated from the date of bud break, to estimate a value for host susceptibility.

10) Equation [19] is used to calculate a value for DPI at this point. The value for DPI is calculated by multiplying the 'Inoculum Potential' value by the value for the interaction between 'Inoculum Release' and 'Leaf wetness/Temperature', and by the value for 'Host Susceptibility'. After the first positive DPI value for a growing season is calculated then a feedback-loop in the model multiplies that value by the 'Inoculum Potential' value to produce an estimate of 'Inoculum Production'. Subsequent calculations of DPI must use the value for 'Inoculum Production'.

11) Disease pressure index values are summed on a real-time basis as they are calculated. Cumulative DPI values are used to assess disease pressure and predict lesion development 5 d in advance using equation [21].

12) The value for cumulative DPI is used to determine if the first control threshold in the model has been exceeded (control thresholds are determined by the user; however, it is suggested that 0 be used for the first and 0.1 be used for the second). If the first control threshold has not been exceeded then the model will continue to calculate and accumulate DPI values. However, if the first control threshold has been exceeded then the model will consider the application and control guidelines for the fungicide Topas 250E, depending on the proximity to fruit harvest.
    a. If fruit harvest has already occurred as indicated by the user, the model will not consider the spray guidelines for Topas 250E, regarding its application before fruit harvest.
    b. If fruit harvest has not yet occurred, then the model must determine if there are more than 38 d until the predicted date of fruit harvest. From the current date, the SASK-Day model is used to calculate the sum of heat units accumulated from the date of bud break and then historical daily SASK-Day heat unit data (calculated from historical temperature data specific to the growing region) are used to predict when fruit harvest (515.9 heat units) will occur (see section 6.4.1 for and example of this procedure).
        1. If there are fewer than 38 d until the predicted date of fruit harvest then the model will not suggest an application of Topas 250E.
        2. If there are more than 38 d until the predicted date of fruit harvest then the model will continue to consider if an application of Topas 250E should be applied.

13) If this will be the first application of Topas 250E then the model will suggest a fungicide application. However, if this is not the first fungicide application then the model must consider if the last fungicide application is still active. In order for the model to determine if the last application of Topas 250E is still active it requires information regarding the number of calendar days since the last fungicide application and an estimate of physiological time since the date of bud break.

    a. If there are ≤ 441.4 SASK-Day heat units accumulated then the model assumes that the last Topas 250E application is active for a period of 14 d.
    b. If there are > 441.4 SASK-Day heat units accumulated then the model will assume that the last Topas 250E application is active for a period of 21d.

14) If the fungicide is no longer active and it is not the first application of Topas 250E then the model will consider if a second control threshold has been exceeded.
    a. If the threshold was exceeded then the model will suggest an application of Topas 250E.
    b. If the threshold was not exceeded then the model will not suggest an application of Topas 250E.

15) Despite the fact that the fungicide may still be active, if the model determines that it is the 39th day before the predicted date of fruit harvest (as calculated using actual and historical SASK-Day heat unit data) then it will consider if a second control threshold is exceeded.
    a. If the second control threshold is exceeded then the model will suggest an application of Topas 250E.
    b. If the second control threshold is not exceeded then the model will not suggest an application of Topas 250E.

16) The user must enter into the model whether or not they applied Topas 250E and the date they applied it.
    a. If Topas 250E was not applied then the model will continue to calculate cumulative DPI.
    b. If Topas 250E was applied then the model counts the number of calendar days since the fungicide application and enters it into clause (12-c-i) in the model, and resets the cumulative DPI value to 0.1. The resetting of the cumulative DPI value results in a lowered 'Inoculum Production' value in the model because of the feedback loop. The model will continue to calculate cumulative DPI after this point.

4. PERSONNEL

R. St-Pierre - 5%
Q. Holtslag - 100%
A. Zatylny - 25%
V. Catinot - 15%

5. EQUIPMENT

Rented: Spectrum AgMet Stations, Alan Splash Spore Trapper, Envirocon growth chambers, Olympus digital camera, Olympus compound microscope, Leitz stereomicroscope, Dell computer and HP printer

6. PROJECT DEVELOPED MATERIALS

Refereed Papers:

Holtslag, Q. A., W.R. Remphrey, W. R., Fernando, W. G. D. Fernando, R.G. St-Pierre, and S. Panicker. 2003. Leaf wetness duration and temperature required for infection of saskatoon leaves by *Entomosporium mespili* under controlled conditions. Can. J. of Plant Pathol. 25: 168-173.

Q. A. Holtslag, W.R. Remphrey, W.G.D. Fernando, R.G. St-Pierre, and R. Dill-Macky. 2004. Estimating inoculum potential relative to infected leaf area and environmental conditions required for stimulating release of *Entomosporium mespili* conidia. Can. J. of Plant Pathol. (in press).

Q.A. Holtslag, W.R. Remphrey, W.G.D. Fernando, R.G. St-Pierre, and G.H.B. Ash. 2004. The development of a dynamic disease-forecasting model to control *Entomosporium mespili* on *Amelanchier alnifolia*. Can. J. of Plant Pathol. (in press).

Ph.D. Thesis:

Holtslag, Q.A. 2003. Modeling Phenology and Entomosporium Leaf and Berry Spot Disease Development in *Amelanchier alnifolia* Nutt., Department of Plant Science, University of Manitoba, Winnipeg, Manitoba.

Abstracts and Papers Presented:

Holtslag, Q. A., Remphrey, W. R., Fernando, W. G. D., Ash, G. H., and St-Pierre, R. G. 2002. Forecasting phenological and disease development for Amelanchier alnifolia. International Society for Horticulture Science, XXVIth Congress and Exhibition Proceedings. TO, Canada. August 2002. S13-P-139, p. 376.

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Holtslag, Q. A., Remphrey, W. R., Fernando, W. G. D., St-Pierre, R. G., Ash, G. H. B., and Dill-Macky, R. 2001. Digital fortune telling: Design and development of a disease forecasting model. Department of Plant Pathology at the University of Minnesota.

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Holtslag, Q.A., W.R. Remphrey, W.G.D. Fernando, R.G. St-Pierre, and G.H.B. Ash. 2001. Digital Fortune Telling: Design and Development of a Disease Forecast Model. Horticulture Production Days, Brandon, MB.

7. PROJECT PHOTOS

Included in Report.

8. ACKNOWLEDGEMENT

Refer to Section 6 above.